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A STUDY OF 212 HOME ACCIDENTS*

JEROME J. DECOSSE, M.D.†
NEW YORK CITY

ACCIDENTS are a problem of increasing importance to modern society. During 1950, there were 90,000 deaths in the United States[1] from this cause. Accidents were the fourth leading cause of death among the total population of Minnesota during 1950, and the most frequent cause of death[2] among persons one to thirty-four years of age, being responsible for more deaths than neoplastic diseases, convulsions and diseases of the central nervous system, heart disease and rheumatic fever, congenital malformations, and pneumonia and influenza combined.[2]

The home is the most frequent site of accident in the United States, 27,500 deaths and 4,100,000 injuries being due to home accidents in the United States in 1950,[1] including 110,000 persons with permanent disabilities. One out of 37 persons was disabled one or more days by injury in the home. In 1950 home accidents constituted two fifths of all accidental deaths in Minnesota, accounting for half the accidental deaths among children under the age of fifteen.[2]

Some of the factors involved are age, sex, disability and alcoholism. Environmental factors such as time, weather, geography and socioeconomic conditions are important.[3] One of the most significant factors is accident proneness. Psychiatrists have related a predisposition to accidents to the total personality structure.[4-6] An accident-prone person is described as one who acts impulsively without deliberation and who harbors an inward rebellion against strict upbringing manifested by resentment against persons in authority, an injury being regarded as an atonement for the guilt of this rebellion. Although this concept is quite popular, there are differences of opinion.[7, 8] Weinermann[8] criticizes an overemphasis on hidden psychologic motivation, with the resultant minimizing of physiologic and environmental factors.

Gordon,[9] who has been the outstanding proponent of the epidemiologic approach to accident prevention points out the steps involved in this approach: determination of the existing status and behavior of the disorder in the community or universe investigated; determination of the causes of the specific disorder; development of specific measures for adequate control; and evaluation of results that come from the recommended principles. The first step, then, toward a program of accident prevention is to obtain detailed information about accidents.

The purpose of this paper is to analyze the interrelated factors in home accidents, with an evaluation of the effect of sex, age, time of day and week, place in the home and activity on incidence; the relative importance of a pre-existing disability, acute alcoholism and accident proneness is also considered. Another objective is the determination of the relation of home accidents to holidays and week ends.

A number of possible approaches to a statistical study are conceivable.[2, 10] One is an organized survey of households in which interviewers ascertain whether the occupants were injured within a specified period. Another is co-operation of the physicians in a selected area in reporting all the accidents of the patients they attend. A third is the incorporation of an accident study into the routine activities of the health department, and a fourth is the study of patients admitted to hospitals with accidental injuries.

METHOD

The last method was chosen for a statistical study of home accidents in Minneapolis. This study was limited to patients who were admitted to the hospital. For each patient admitted, many minor injuries were treated in the emergency room, but since only the more serious accidents cause an economic loss and a temporary or permanent disability, they were considered more significant from the point of view of preventive medicine. This study dealt with "disabling accidents," or those preventing a patient from attending to his ordinary occupations on the day after the accident.[1]

The charts of all the patients with accidents admitted to the Minneapolis General Hospital from January 1, 1950, through December 31, 1950, were examined. This institution was chosen because, as a municipal hospital, it afforded a large number of accidents for study, the accidents were accurately located (this hospital maintains the Standard

*From the School of Public Health, University of Minnesota.
†Intern in surgery, Roosevelt Hospital; formerly, student, University of Minnesota Medical School.

Nomenclature of Diseases) and complete histories taken by the resident, the intern and often the medical student were available.

The charts were located through the disease index. The accidents obtained in this way were verified by a check of the admission cards for 1950, most of which contained an admission diagnosis.

The cases were logically divided into adult and pediatric age groups, for there was a variation between the age groups not only in the kinds of accidents but also in the causal factors. Tabulations were kept of the place and nature of the injury,

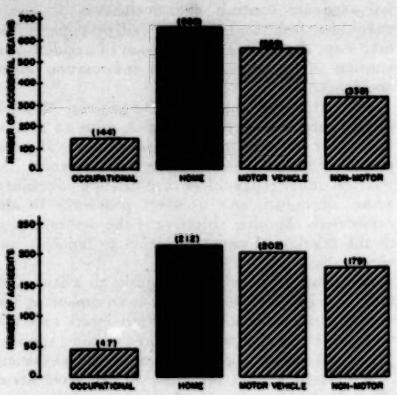

FIGURE 1. *Accidental Deaths Reported in Minnesota in 1950 (Top) Compared with Admissions to Minneapolis General Hospital Because of Accidents in the Same Year (Bottom).*

sex, age, date, time, activity, existing disability, history of previous accidents and the presence of acute alcoholism.

Certain limitations were inherent in the method used. The patients do not represent a universe but a restricted group. The public character of the Minneapolis General Hospital may alter the number and kinds of accidents. The study may reflect the economic level particularly served by the hospital. In occasional cases the investigated material was not available on the chart.

RESULTS

During 1950 admissions to the Minneapolis General Hospital totaled 8760, 640 of which were due to accidents. The accidents were grouped and compared with mortality statistics from accidents in the entire state of Minnesota during 1950[1] (Fig. 1). The similarity between the fatal and nonfatal injuries is apparent. Home accidents accounted for 212 cases, or 32 per cent of all patients admitted because of accidents.

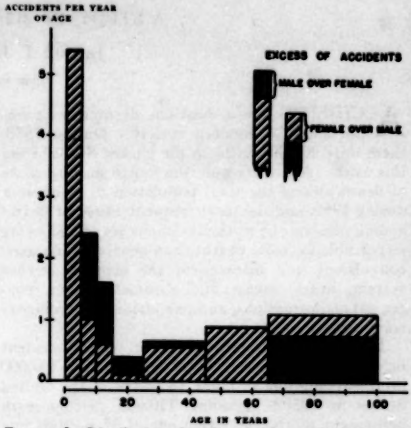

FIGURE 2. *Distribution According to Sex and Age among Patients Admitted to Minneapolis General Hospital Because of Accidents in 1950.*

There were 69 deaths among these patients, 21, or 30 per cent, being due to home accidents. It is of interest that during August and September, although 38 patients were admitted to the hospital after home accidents, 368 patients were treated in the emergency room after such an accident. The majority of the latter group suffered from minor lacerations.

TABLE 1. *Distribution According to Sex and Type of Accident.*

SEX	FALLS		BURNS		FOREIGN BODIES	CONTUSIONS, ETC.	POISONING	MISCELLANEOUS	TOTALS
	ADULTS	CHILDREN	ADULTS	CHILDREN	NO. OF CASES	NO. OF CASES	NO. OF CASES	NO. OF CASES	
Male	44	9	6	14	5	11	15	5	108
Female	50	8	5	6	11	3	15	5	104

The distribution by sex and kind of home accident is presented in Table 1. There was little sex difference in the total number of accidents, although there was a slight predominance of boys in the pediatric range, particularly in burns and contusions, lacerations and abrasions. Figure 2 shows a slight

TABLE 2. *Location of Home Accidents.*

LOCATION		No. OF CASES
Kitchen		45
Stairs		43
Inside	25	
Outside	14	
Stairs only	4	
Bedroom		34
Yard		26
Parlor		13
Bathroom		10
Porch		4
Not stated		37
Total		212

male preponderance among patients five to forty-four years of age and a female predominance in the age group over sixty-five years. Over 56 per cent of the total occurred in the two extreme age groups — namely, under five years and over sixty-five years.

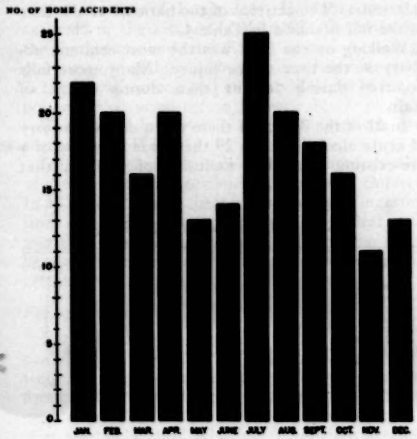

FIGURE 3. *Incidence by Months of Home Accidents among Patients Admitted to Minneapolis General Hospital in 1950.*

The kitchen was the most common place for an accident in the home, followed in order by the stairs, the bedroom and the yard (Table 2). In other studies of fatal home accidents, it has been shown that the bedroom, the yard and the kitchen were, respectively, involved.[11] The bathroom, which has received a large amount of publicity, was relatively unimportant. These data are in contrast to those of Roberts,[12] who showed that 54.1 per cent occurred outside the house. Here, only 24 per cent could be considered as occurring outside the house.

A study of the month in which the accident occurred is presented in Figure 3. There seems to be a relatively even distribution. The slight decline in November and December could not be explained on any other basis than errors of random sampling. The high incidence in July is attributed to an outbreak of children's accidents. A recent study by Roberts[12] demonstrated a statistically insignificant seasonal variation in incidence. In her studies, the

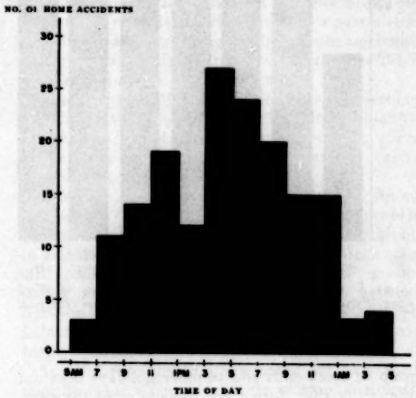

FIGURE 4. *Occurrence of Home Accidents by Time of Day among Patients Admitted to Minneapolis General Hospital in 1950.*

incidence in January was almost twice that in October, the month with the lowest number of home accidents.

Figure 4 shows a study of the time of day in which the accident occurred. It is noteworthy that there were relatively few accidents in the morning, and that as the day progressed the number increased, with peaks seemingly at the times of greatest activity. Over 64 per cent occurred after 2 p.m. If this twelve-hour interval is divided into three four-hour periods, and the observed and calculated data compared, the probability (P_{x^2}) that the results are due to chance alone is 0.01.

Figure 5 presents a comparison of the number of home accidents according to the day of the week. The largest number occurred on Monday and Saturday, the days of possibly greatest activity, almost twice as many being reported on Monday as on Sunday, the day with the lowest number of

home injuries. The probability that these results were due to errors of random sampling is $0.24 < (P\chi^2) < 0.32$.

A comparison of the frequency in adults on week ends and the remainder of the week shows no significant variation. The incidence in the pediatric

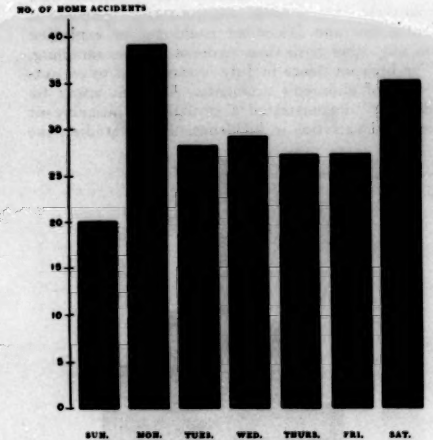

FIGURE 5. *Occurrence of Home Accidents by Day of Week among Patients Admitted to Minneapolis General Hospital in 1950.*

group on week ends and summer holidays does not differ from that during the remainder of the year.

The numerical frequency of the kinds of home accidents is presented in Table 3.

These findings generally coincide with those in other studies of nonfatal home accidents.[13-15] There

TABLE 3. *Types of Home Accidents.*

ACCIDENT		No. OF CASES
Fall		111
Adults	94	
Children	17	
Burn		31
Adults	11	
Children	20	
Poisoning		30
Adults	3	
Children	27	
Foreign body		16
Contusion, laceration and abrasion		14
Gunshot wound		3
Miscellaneous		7
Total		212

is an increased incidence of poisonings and a decreased frequency of contusions, lacerations and abrasions in this study.

Falls

Falls were numerically the most frequent of disabling injuries in this study, as well as in all other surveys. Falls were most frequent in the two extreme age limits, particularly in elderly persons. Elderly women were most susceptible to falls. There was no predilection for the time of year. Falls tended to

TABLE 4. *Activity of Patient at Time of Fall.*

ACTIVITY	No. OF CASES
Walking on level	25
Walking downstairs	22
Standing	10
Walking upstairs	9
Walking on stairs	8
Arising	8
On ladder	4
Miscellaneous	8
Total	94

increase in the afternoon and evening. By far the most common disabling result was a fracture in and about the hip joint. The inside stairs were the most likely place in the home for the fall to occur, being followed in frequency by the bedroom and the outside stairs. The activity of the patients at the time of the fall is shown in Table 4.

Walking on the level was the most common activity at the time of the injury. Many more falls occurred during descent than during ascent of stairs.

In 21 of the 94 adults there was a definite history of acute alcoholism. In 29 there was a history of a pre-existing disability, exclusive of senility, that

TABLE 5. *Agents Involved in Poisoning.*

AGENT	No. OF CASES
Kerosene and fuel oil	12
Turpentine	3
Phenolphthalein (Ex-lax)	2
Barbiturates	2
Digitalis (adults)	2
Camphorated oil	2
Miscellaneous (opium, furniture polish, arsenic, Ergot, strychnine and argyrol [adults])	7
Total	30

directly predisposed the patient to the accident. Examples were cerebrovascular accidents, poor vision or blindness, seizure and syncope. In only 7 of the 94 adults was a history of 2 or more previous accidents elicited.

Falls in children showed a predilection for June and July, the most likely place for the fall to occur being the yard. All but 2 of the children were in some form of play at the time of the fall. In 5 of the 17 injuries, an older person was directly responsible for the accident.

Burns

Burns in adults occurred most frequently in the bedroom or the kitchen, whereas those in children most often took place in the kitchen or, secondly, in the yard. In the pediatric age groups, boys outnumbered girls 14 to 6. Fourteen of the 20 burns in this group occurred in children four years of age or under. Seven of the burns were scalds, and 13 were due to flame. The most common story was the spilling of a hot liquid, such as coffee, boiling water or milk, from a pan on the stove. At the ages below two years, upsetting of hot fluids was more common, whereas in older children burning of clothing was more frequent.

Poisoning

Poisoning in adults was almost always deliberate. Since suicide attempts were not considered accidental but deliberate, only 3 cases of adult poisoning are included. These were associated with accidental overdosage of some medication.

Only 5 of the 27 children who were poisoned were over two years of age, 15 of the 27 cases occurring between sixteen and twenty-four months of age. Most of the children were engaged in some form of play at the time of the accident. Half the poisonings occurred in the kitchen, and half in the bedroom, the bathroom and the living room. Accidental poisonings in children showed a tendency to occur in the late afternoon and evening. Only 9 of the 27 poisonings occurred before 3 p.m.

The agents involved in the poisonings are presented in Table 5.

It is interesting that some older person was present at the time of the ingestion of poison by 9 children. In 13 of the 21 cases there was a history of ingestion from a previously open container. A typical story was that of an eighteen-month old boy who drank kerosene from an open can lying on the floor in the kitchen beside the stove.

Foreign Bodies

Foreign bodies were divided evenly between the pediatric and the adult age groups. Chicken bones were the most frequent foreign body. Half the foreign bodies were in the esophagus. Only 1 case was in a patient with a pre-existing disability, and that was in a child who had a repaired tracheoesophageal fistula.

Contusions, Lacerations and Abrasions

This type of accident showed a predilection for male adults of the younger age group. Tendon lacerations constituted the most frequent cause considered serious enough for hospital admission.

Discussion

Certain aspects of the results deserve additional comment. The contrast between the data of Roberts[12] and those of this study in the frequency of accidents in the yard may be related to the colder weather in Minnesota. In Massachusetts the mean temperature is higher, and consequently there is more activity outdoors.

The increasing number of accidents during the course of the day may have been related to increasing fatigue. A fatigue factor, demonstrated in industrial and automobile accidents, is suggested to explain this variation in home accidents. In the pediatric age group, it cannot be ascertained whether the child or the parent was tired.

The large number of patients with concomitant acute alcoholism probably reflects in part the municipal nature of the Minneapolis General Hospital. A comparison of the number of patients with acute alcoholism, pre-existing disability and a past history of previous accidents suggests that acute alcoholism and pre-existing disability are important factors in the background of home accidents.

The frequency of kerosene and fuel-oil poisonings may be related to the economic level from which the patients come and to the latent respiratory effects of kerosene poisoning, which necessitate careful observation.

From the foregoing data the importance of home accidents is obvious. The magnitude of the problem deserves the recognition and support of many groups in addition to health departments and safety councils. Medical societies, parent-teacher groups, police and fire departments, welfare agencies, building inspectors, insurance companies and even industry have a responsibility in the prevention of home accidents.

Specific recommendations based on this study may be made. The importance of emphasis on the preschool child is apparent. The older woman and the person with pre-existing disability should also be given attention. The use of afternoon rest periods in the older person may be a preventive measure for home accidents. The large number of falls that occurred while the patients were around the bed suggest the placing of chairs, dressers or railings beside the bed of the aged and disabled to give them support.

Many of the preventive efforts for poisonings at present apply to adults. This survey suggests the infrequency of accidental adult poisonings and the need for emphasis in the child under two years of age. From the large number of kerosene poisonings, continued emphasis on the placing of these containers beyond the reach of children is necessary.

The need for further work in statistical studies of home accidents is apparent. The variation of accidents by day and month, the effect of the time of day on poisonings and the relation of sex in pediatric accidents deserve the collection and evaluation of more data.

Only a part of the history was given in the records. A further study might seek more detailed knowl-

edge of the situation in which the accident occurred. The presence of emotional tension or fatigue, the lighting conditions and the presence of hazards such as loose rugs and a lack of guard railings are among the questions that might be asked.

Many of the limitations of this study could be overcome by a more ideal approach. The co-operation of the members of the county medical society and public-health nurses might be obtained. If all the home accidents serious enough to require medical attention were studied carefully, a valuable contribution could be made.

Conclusions

There is a significant variation in the number of accidents during the day, with an increasing frequency as the day progresses. There is suggestive evidence that this variation is related to a fatigue factor.

There is suggestive evidence that existing disability and acute alcoholism are more important factors in home accidents than accident proneness.

The preschool child, the older woman, the aged and the patient with a pre-existing disability should be given particular attention in preventive efforts.

The prevention of accidental poisonings should be directed at the child under two years of age.

The incidence of home accidents on week ends and holidays does not vary significantly in either the pediatric or the adult age group.

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GASTRITIS AND GASTROSCOPIC BIOPSY*

Sydney Selesnick, M.D.,† and Edward D. Kinsella, M.D.‡

Newington and New Haven, Connecticut

In 1937 Palmer[1] hailed Schindler's flexible gastroscope as the instrument that had finally brought the element of objectivity into the diagnosis of disease of the gastric mucosa. In 1947 Schindler[2] himself expressed delight at the general acceptance of his diagnostic instrument only fifteen years after its introduction. Benedict,[3] in 1948, devised the operating gastroscope; as a result of his experience with biopsies of gastric mucosa obtained with this instrument, he challenges the adequacy of conventional gastroscopy as a diagnostic method. He believes that no gastroscopic examination is complete unless biopsy under direct vision is available.[4] The experience of others, using this operating gastroscope, appears to support his contention.[5]

This is a serious indictment of the adequacy of conventional gastroscopy, especially in the diagnosis of gastritis. The texts by Schindler[2] and Palmer,[6] which are the basis of the interpretation of disease of the gastric mucosa in this country, followed extensive experience with conventional gastroscopy. To be sure, post-mortem and surgical specimens were used to formulate diagnostic criteria, but these are admittedly not satisfactory for the interruption of disease in specific localized areas of mucosa seen in vivo.

The purpose of this study is to examine the validity of Benedict's belief that no gastroscopic examination is complete unless biopsy under direct vision is available. The records of 50 patients who had gastroscopic examinations, including mucosal biopsy obtained with the Benedict operating gastroscope, were examined. Only gastric mucosas that were interpreted as appearing normal or manifesting some form of gastritis were reviewed. In every case the gastroscopist considered the patient co-operative and the gastric mucosa adequately visualized, within the recognized limitations of gastroscopy, and the pathologist accepted the tissue specimen as adequate for histologic study. Almost all the patients were examined with both conventional and operating gastroscopes. There were 56 gastroscopic examinations with mucosal biopsy in 50 patients. All the patients had upper gastrointestinal complaints.

Results

The gastroscopist considered that in 23 of the 56 examinations the gastric mucosa appeared normal. The pathologist concurred in only 14 (60 per cent)

*From the Veterans Administration Hospital, Newington, and Yale University School of Medicine, New Haven, Connecticut.

†Assistant professor of clinical medicine, Yale University School of Medicine; gastroenterologist, Veterans Administration Hospital, Newington.

‡Instructor in medicine, St. Louis University School of Medicine, St. Louis, Missouri; formerly, fellow in gastroenterology, Veterans Administration Hospital, Newington, Connecticut.

of these and in 9 (40 per cent) interpreted the mucosa as showing evidence of gastritis. In 33 examinations the gastroscopist found evidence of gastritis, and the pathologist concurred in 30, or 91 per cent, of the histologic specimens obtained from the suspect mucosa, disagreeing in 3 (9 per cent). Conversely, of 17 tissue specimens considered by the pathologist to be normal, the gastroscopist agreed in 14 (82 per cent) and disagreed in 3 (18 per cent), whereas in 39 that the pathologist considered to show gastritis the gastroscopist concurred in 30, or 77 per cent, disagreeing in 9, or 23 per cent. Obviously, the greatest disparity existed in the gastroscopist's interpretation of normal mucosa, with which the pathologist was in agreement in only 60 per cent. Since there were but 23 examinations in this group, additional records were reviewed to include all patients with grossly normal mucosa who had had mucosal biopsies. Forty-one were found whose mucosa appeared normal to the gastroscopist. In this group, almost double the first, the pathologist agreed that the mucosa was histologically normal in only 23 patients (56 per cent) but found gastritis present in 18 patients (44 per cent). This reinforces the observation that the greatest disparity between pathologist and gastroscopist was in the interpretation of the normal. From this study it seems that when the gastroscopist recognizes gastric mucosal disease a biopsy will usually confirm his findings (91 per cent). However, when the mucosa appears grossly normal, there is hardly better than an even chance that a biopsy will be confirmatory (56 per cent).

Discussion

What can be learned from this study that might explain why the gastroscopist and pathologist are so often at variance? Benedict[7] asks, "If symptoms consistent with gastritis are present and the gross gastroscopic picture is definitely that of gastritis, but the biopsy shows a normal gastric mucosa, should one rely on the gastroscopic picture or on the pathologist's report?" He provides the following answer: "Since positive evidence is better than negative evidence, one relies on the observation of a trained gastroscopist, discounting somewhat the pathologist's report of negative findings as being due to an error in sampling — meaning that the biopsy specimen may have been taken from a relatively normal area in the midst of other areas which would have revealed gastritis." Such a formulation entertains certain objections. It seems fair to believe that all has not yet been learned about the gastroscopic picture of the mucosa in health and disease. Granting this, it may be that gastroscopists have been trained to recognize gastritis where it does not, in fact, exist and not to recognize it where it does. Although a biopsy specimen might be taken from a relatively normal area in the midst of other areas that would have revealed gastritis, this seems hardly likely. Such sampling errors could occur commonly with the virtually blind methods of suction biopsy but should be exceptional when tissue is obtained under direct vision.[8, 9] To reject the validity of a negative biopsy, obtained under direct vision, is tantamount to denying that the gastroscopist's observations may be incorrect. The question of the recognition of areae gastricae* is pertinent. Most gastroscopists no longer vex themselves about whether a pebbled patch of mucosa represents chronic hypertrophic gastritis or the normal mammillations of areae gastricae, but there are still a few who do. This controversy is not altogether settled. Mucosal biopsy under direct vision with the operating gastroscope can shed some light on this problem.

In this study the gastroscopist considered 33 mucosas to show gastritis. The pathologist concurred in 91 per cent of these, but in 3 cases (9 per cent) he found the tissue to be histologically normal. Each of these three mucosas was said by the gastroscopist to have manifested chronic hypertrophic gastritis (Table 1).

Table 1. *Comparison of Gastroscopic and Pathological Diagnosis of 56 Gastric Mucosas Biopsied under Direct Vision.*

No. of Patients	Gastroscopic Diagnosis	Pathological Diagnosis
3	Hypertrophic gastritis	Normal mucosa
5	Hypertrophic gastritis	Chronic gastritis
1	Hypertrophic gastritis	Acute and chronic gastritis
6	Superficial gastritis	Acute and chronic gastritis
2	Superficial gastritis	Chronic gastritis
3	Atrophic gastritis	Chronic gastritis
1	Atrophic gastritis	Chronic gastritis, with intestinalization*
1	Atrophic gastritis	Acute and chronic gastritis
4	Mixed gastritis	Acute and chronic gastritis
2	Mixed gastritis	Chronic gastritis, with intestinalization
1	Mixed gastritis	Chronic gastritis
2	Gastritis (unclassified)	Chronic gastritis
1	Gastritis (unclassified)	Acute gastritis
1	Gastritis (unclassified)	Acute and chronic gastritis, with intestinalization
14	Normal mucosa	Normal mucosa
5	Normal mucosa	Chronic gastritis
3	Normal mucosa	Acute and chronic gastritis
1	Normal mucosa	Chronic gastritis, with intestinalization

*Replacement of destroyed gastric mucosa by an intestinal type of columnar epithelium.

The first patient (Case 52,153), who was examined on December 22, 1949, was said to have a patch of hypertrophic gastritis involving a fold high on the anterior wall of the proximal half of the stomach. On December 29 re-examination was performed to obtain tissue for biopsy. The fold described above was not seen, but the posterior-wall mucosa was said to have an area of hypertrophic pebbling. Tissue was taken from this area. The pathologist reported normal-appearing gastric mucosa. On February 3, 1950, the patient was again examined, and no abnormalities were noted.

The second patient (Case 46,977) was first gastroscoped in December, 1948. A postgastrectomy stomach was seen with

*These are normal anatomic features commonly seen on the mucosal surface of the fresh stomach. They are numerous small, slightly elevated polygonal areas, 1 to 6 mm. in diameter, separated by linear depressions giving a mammillated appearance to the mucosal surface. Palmer[4] states that these structures are a feature of the dead, not the living, stomach and that they should not cause confusion in the diagnosis of chronic hypertrophic gastritis.

a normally functioning stoma and normal mucosa. He was again examined in April, 1950, for recurrent ulcer-like symptoms. On the anterior-wall side of the lesser curvature near the stoma a patch of lustreless, pebbled-appearing mucosa, the size of a half-dollar, was seen. The pathologist's report of a biopsy taken from this area was gastric mucosa without evidence of inflammation.

The third patient (Case 56,838) was described by the roentgenologist as having an ulcer pocket on the lesser curvature of pars media with a broad, stiffened rugal fold near the ulcer suggesting cancer or gastritis. The first gastroscopy, done on January 31, 1951, disclosed a diffusely pebbled, lusterless mucosa. On the lesser curvature of pars media the mucosa was heaped up to form a large prominence covered by "cobbled" mucosa. The ulcer was not seen. A diagnosis of chronic hypertrophic gastritis was made. On February 5 tissue for biopsy was taken. The specimen was inadequate for diagnosis. On February 26 tissue was again obtained "from heaped up lusterless mucosa, cobbled, and mottled red and gray." The pathologist described a specimen showing well differentiated gastric mucosa that had the usual cell types, with evidence of active secretion. The lamina propria contained only a small number of plasma cells and eosinophils. The diagnosis was normal gastric mucosa or possibly mild gastritis.

Since there was no reason to believe that there had been an error in sampling, the pathologist's report was accepted as the correct one. Therefore, it was deemed necessary to revise our criteria for the diagnosis of chronic hypertrophic gastritis. Pebbling and diminution of luster were no longer accepted as adequate evidence on which to base such a diagnosis. It was recognized that any mechanism, physiologic or pathologic, that disrupted the usually smooth surface of normal mucosa (and that might be referred to as pebbling, cobbling, granularity, or mammillation) would alter the reflection of the light back to the eye of the observer so that the mucosa would appear abnormal. Therefore, individual mammillations as well as the involved area as a whole were examined. It was first determined that the area in question was properly illuminated and did not lie in partial shadow. If the mammillations reflected highlights and the texture of the mucosa at these points appeared normal, and no associated abnormalities were seen, the suspected area was considered normal. On the basis of these criteria, 4 subsequent gastroscopic studies that would previously have been interpreted as representing chronic hypertrophic gastritis were reported as normal mucosa with mammillation. The pathologist also reported normal mucosa in all 4. Whether these represent areae gastricae cannot be said for certain, but it does seem that physiologic mechanisms, in some persons, can produce a gastroscopic picture resembling chronic hypertrophic gastritis. It is interesting that the pathologist reported normal mucosa in 2 of 20 patients considered by Palmer[10] to have chronic hypertrophic gastritis. Benedict[7] also reported a normal biopsy in a patient considered by him to have chronic hypertrophic gastritis.

In 9 of 23 cases in which the gastroscopist reported normal mucosa the biopsy gave evidence of gastritis (Table 1). It is well recognized that microscopical changes precede the gross changes of an inflammatory reaction and so may account for some of these errors. A typical case is as follows:

Gastroscopy, performed in this patient (Case 55,702) on October 16, 1950, described excellent visualization of normal mucosa to the pyloric orifice. Biopsy from normal-appearing mucosa of the lesser curvature proximal to the angulus was interpreted by the pathologist as acute and chronic gastritis. Repeat gastroscopy on January 18, 1951, disclosed moist, succulent mucosa with thick, full, rounded rugal folds and scattered white, adherent exudate. The diagnosis was superficial gastritis. The pathologist reported chronic gastritis. A third examination, on March 5, showed an excessively moist and succulent but not injected mucosa, scattered, adherent, thick, white material and pebbling along the greater curvature. The diagnosis was mixed gastritis. Biopsy from a pebbled area on the greater curvature opposite the angulus gave evidence of chronic gastritis.

Palmer[10] has already pointed out that the mucosa in histologic acute focal gastritis frequently appears grossly normal. He correctly holds that failure to apply proper objectivity, failure to limit audience participation, failure to do serial examinations and too close adherence to classic gastroscopic criteria for diagnosis are causes of error in interpretation. One may also add an intangible factor, the human equation, which interferes with individual objectivity from time to time.

Summary

A review of 56 satisfactory gastroscopic examinations, including biopsy of gastric mucosa under direct vision, in 50 patients considered to have either grossly normal mucosa or gastritis appears to validate Benedict's belief that gastroscopy is not complete without mucosal biopsy under direct vision. This seems to be especially true in cases of normal-appearing mucosa and those considered to manifest chronic hypertrophic gastritis. It is proposed that physiologic alterations, possibly areae gastricae, may simulate chronic hypertrophic gastritis.

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THE EFFECT OF CORTISONE THERAPY ON THE INCIDENCE OF RHEUMATIC HEART DISEASE*

ARNOLD L. JOHNSON, M.D., C.M.,† AND CHARLOTTE FERENCZ, M.D., C.M.‡

MONTREAL, CANADA

THE purpose of this report is to attempt to assess the effect of cortisone in rheumatic heart disease by consideration of a group of 100 patients treated with cortisone and, for purposes of comparison, a group of 80 patients cared for under similar circumstances, before cortisone was utilized in this hospital. Since the original contribution of Hench and his co-workers[1] pointed the way to the use of cortisone in the treatment of rheumatic fever, there has been a constantly growing list of valuable communications reporting experience with series of patients of variable size and from slightly different points of view. This literature has been referred to sufficiently in many papers. There has been a growing impression that the incidence of heart involvement is not being reduced by cortisone and adrenocorticotrophin (ACTH) therapy; it is also noted that, in the treatment of the patient acutely ill with rheumatic pancarditis, the relief afforded by these hormonal agents has often been dramatic but that failures of therapy are not infrequent. No statistical evidence has yet been presented by which one may determine the effect of cortisone and ACTH on the incidence of rheumatic heart disease, and this paper is concerned with that problem.

Two groups of patients have been established: the cortisone group and the precortisone group.

The former comprised 100 children between the ages of three and fourteen years admitted to the Children's Memorial Hospital between March, 1950, and June, 1952. The patients all had active rheumatic fever and fulfilled the diagnostic criteria recommended by Jones.[2] They were treated on a fairly uniform schedule of therapy. Cortisone was given intramuscularly to 56 patients. The usual regimen was 200 mg. for two days and 100 to 150 mg. daily thereafter for twenty-one days. Ten patients received the hormone for thirty-one to fifty days. Cortisone was given orally to 38 patients; the majority received 200 mg. for two days and 100 to 150 mg. daily for twenty-eight days, with the exception of 6 patients treated for forty to eighty-four days. Six patients received ACTH intramuscularly in daily dosages varying between 40 and 60 mg. for seven to forty days. Various clinical and laboratory observations were made before, during and after therapy, and these data have been reported for the first 39 patients.[3] The present report is concerned only with the evaluation of the cardiac status by clinical, electrocardiographic and fluoroscopic means.

The precortisone group was composed of patients, also under the care of the Department of Cardiology in this hospital, admitted before March, 1950, when our first patient was treated with cortisone. Patients were selected by the same criteria as those in the cortisone-treated group from a chronologic list. Thus, if one works backward from this list, patients who would have been treated with cortisone if they had presented themselves after March, 1950, were selected for inclusion in the precortisone group. In this way, 80 patients between the ages of three and fourteen were chosen between February, 1950, and January, 1947. Management of this group consisted of bed rest and symptomatic

TABLE 1. *Distribution of Patients According to Cardiac Manifestations During "Treated" Episode.*

GROUP	NO. OF CASES	NO MANIFESTATIONS	MINIMAL MANIFESTATIONS	MODERATE MANIFESTATIONS	SEVERE MANIFESTATIONS	PRIOR RHEUMATIC HEART DISEASE
		%	%	%	%	%
Precortisone ..	80	26.2	31.3	11.25	13.75	17.50
Cortisone	100	30.0	25.0	20.0	12.0	13.0

TABLE 2. *Incidence of Cardiac Manifestations.*

GROUP	CARDIAC MANIFESTATIONS DURING "TREATED" EPISODE	CARDIAC MANIFESTATIONS ON DISCHARGE FROM HOSPITAL OR DEATH
	%	%
Precortisone	73.8	42.55
Cortisone	70.0	43.0

*From the Children's Memorial Hospital and the Department of Paediatrics, McGill University Faculty of Medicine.

The cortisone used in this study was supplied in part by the National Research Council of Canada and in part by Merck and Company, Limited, through the kindness of Dr. J. H. Laurie, medical director.

†Director, Department of Cardiology, Children's Memorial Hospital.

‡Formerly, research fellow, Canadian Arthritis and Rheumatism Society.

therapy. Many of these patients had received salicylate therapy for the relief of joint pain, but in only a few cases had this therapy been continued for more than seven to ten days.

Patients in each group were subdivided according to our assessment of the severity of cardiac manifestations presented during the course of the active rheumatic episode for which they were included in the series. This is referred to as the "treated" episode. The division, according to the criteria described below, is, of course, an artificial one, but because the selection of each group of patients, precortisone and cortisone, has been made by the same two observers, the errors inherent in such a division apply to both series. The value of the grouping lies in the fact that an approximate idea of the general severity of the cases being treated may thereby be obtained.

This grouping, according to the most marked manifestations of heart disease during the "treated" episode, places patients in one of five categories: no cardiac manifestations — no clinical, x-ray or electrocardiographic evidence of heart disease; minimal manifestations — a slight apical systolic murmur (Grade 1 or Grade 2 in Levine's[4] classification) or electrocardiographic changes or both; moderate manifestations — apical systolic murmur of at least Grade 3 intensity, any diastolic murmur and some cardiac enlargement; severe manifestations — any of the preceding manifestations and, in addition, marked cardiac enlargement, pericardial involvement, congestive heart failure or pulmonary edema; and prior rheumatic heart disease — patients who have had heart disease before the "treated" episode must be considered separately because of the difficulty in assessing any form of therapy in them. Patients who died are included in one or the other of the last two groups.

Table 1 indicates that, over the five-and-a-half-year period from which these two series of patients were drawn, the severity of the disease as judged by the cardiac manifestations was similar. A χ^2 test shows that there is no statistical difference between the two distributions ($\chi^2 = 3.74$, $P = 0.3 - 0.5$).* Since the general approach to the disease, the medical observation, the nursing care and the symptomatic therapy were similar, it appears to us that the comparison of these two groups of patients is of value in the assessment of cortisone therapy.

Table 2 summarizes the incidence of cardiac manifestations in each group during the "treated" episode and on discharge from the hospital. With a few exceptions, patients were considered fit for discharge when all the usually accepted evidences of rheumatic activity, including the sedimentation rate, had subsided. The striking similarity of the figures appears to indicate not only that the incidence of cardiac manifestations in the two groups was similar but also that the persistence of heart involvement in the period of hospitalization was the same.

Table 3 shows the incidence of the persistence of heart disease in relation to the severity of the illness in each group. The figures are derived from those shown in Table 1 with the further breakdown into patients who, on discharge from the hospital, had residual heart disease and those who did not have any evidence of heart involvement. It is evident that there is no significant difference in the two series in the incidence of heart disease after the "treated" episode. The one possible exception to this statement pertains to mortality. The lower death rate in the cortisone-treated group is not statistically significant ($P > 0.05$).[5] However, in view of the dramatic improvement in many severely ill patients, it remains possible that the incidence of death from acute rheumatic fever was reduced by cortisone. This effect may be proved in larger series of cases.

TABLE 3. *Distribution of Patients According to Cardiac Manifestations on Discharge from the Hospital.*

GROUP	NO. OF CASES	NO CARDIAC MANIFESTATIONS	MINIMAL CARDIAC MANIFESTATIONS		MODERATE CARDIAC MANIFESTATIONS		SEVERE CARDIAC MANIFESTATIONS			PRIOR RHEUMATIC HEART DISEASE		
			NO RESIDUAL DISEASE	RESIDUAL DISEASE PRESENT	NO RESIDUAL DISEASE	RESIDUAL DISEASE PRESENT	NO RESIDUAL DISEASE	RESIDUAL DISEASE PRESENT	DEATH	NO CHANGE	INCREASE	DEATH
		%	%	%	%	%	%	%	%	%	%	%
Precortisone	80	26.2	27.5	3.8	1.25	10	2.5	7.5	3.75	15	1.25	1.25
Cortisone	100	30.0	19.0	6.0	4.0	16	4.0	7.0	1.0	11	1.0	1.0

TABLE 4. *Residual Cardiac Involvement.*

GROUP	PATIENTS WITH CARDIAC MANIFESTATIONS DURING TREATED EPISODE	CASES OF HEART DISEASE ON DISCHARGE FROM HOSPITAL	
		NO.	PERCENTAGE
Precortisone	45	21	46.6
Cortisone	57	31	54.4

*We are indebted to Dr. Clarke Fraser, assistant professor, Department of Genetics, McGill University, for the statistical analyses in this paper.

Tables 4 and 5 present a study of the groups that had cardiac manifestations for the first time during the treated episode. Therefore, patients with no cardiac manifestations and those with prior heart disease are excluded from these tables. Table 4 shows that, of the 45 patients with cardiac manifestations during the treated episode in the precortisone group, 21, or 46 per cent, were left with residual heart involvement, as compared with 54 per cent in the cortisone group. There thus appears to be no difference between the two groups in this regard ($x^2 = 0.5996$, $P = 0.3 - 0.5$). A more detailed consideration of the same figures is presented in Table 5. Again, the figures are similar for the two groups except for the apparent reduction in mortality noted above.

TABLE 5. *Incidence of Residual Cardiac Involvement According to Severity of Disease during Treatment.**

GROUP	MINIMAL CARDIAC MANIFESTATIONS		MODERATE CARDIAC MANIFESTATIONS		SEVERE CARDIAC MANIFESTATIONS		TOTALS	MORTALITY
	TOTAL CASES	CASES OF RESIDUAL HEART DISEASE	TOTAL CASES	CASES OF RESIDUAL HEART DISEASE	TOTAL CASES	CASES OF RESIDUAL HEART DISEASE	DEATHS	
								%
Precortisone	25	3 (12%)	9	8 (89%)	11	6 (54.5%)	3 (27.2%)	45	6.7
Cortisone	25	6 (24%)	20	16 (80%)	12	7 (58.2%)	1 (8.35%)	57	1.75

*Breakdown of data in Table 4.

During the course of cortisone therapy one becomes so impressed with the appearance of well-being of the patients and with the marked clinical improvement in some children with acute carditis that the similarity of the incidence of heart disease in the two series of cases comes as a surprise. The incidence of cardiac involvement in the cortisone-treated group was similar to that noted in the precortisone group. If cortisone, in the dosage and for the period of administration employed, had had an influence in reducing the occurrence of rheumatic heart disease, this fact should have become apparent in this study.

SUMMARY

The incidence of rheumatic heart disease in 100 children treated with cortisone and, in a few cases, adrenocorticotrophin is compared with that in 80 patients observed before the use of cortisone. Each series of patients was grouped according to the clinical evidence of the severity of cardiac involvement, and, in view of the similarity of this distribution, it appeared profitable to make a comparison between the two series. The data indicate that there is no significant difference in the incidence of heart disease after the treated episode in the cortisone and precortisone series. Clinical observation suggests the value of cortisone therapy in the extremely sick patient with rheumatic carditis. The possibility that the mortality of acute rheumatic fever will be reduced remains to be assessed.

These patients were treated in co-operation with the Sub-Committee on Cortisone and ACTH of the Children's Memorial Hospital (Drs. Alton Goldbloom, Alan Ross, Eleanor Harpur and Frances McCall, chairman).

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THE USE OF THE ABSOLUTE EOSINOPHIL COUNT IN THE DIAGNOSIS OF NEOPLASMS

Preliminary Report

ROGER C. MURRAY, M.D.*

HANOVER, NEW HAMPSHIRE

SINCE differentiation of a necrotic neoplasm from an infectious process is often difficult, any diagnostic technics that might help should be welcome. The observation by previous workers[1, 2] of an increase in the number of white blood cells accompanied by a relative eosinophilia in patients with various neoplasms suggested that necrotizing tumors stimulate all elements of the leukocyte series, and not solely the neutrophils. If an absolute eosinophilia occurs with necrotizing tumors but not with simple infectious processes, the eosinophil count should prove to be a valuable test in differential diagnosis. This study was undertaken to determine the worth of that possibility.

Historical Review

Eosinophilia in cancer was first reported by Rheinbach[3] in 1893. Kappis,[4] in 1907, and Dunger,[5] in 1910, attributed it to necrosis of tumor tissue, the products of protein breakdown causing an eosinophilotactic response. Others ascribed the eosinophilia to the stimulating effect of bone-marrow metastasis. In 1935 Paviot and his co-workers[6] suggested that the eosinophils originated from the connective tissue surrounding the tumor since local tissue eosinophilia is known to exist around malignant tumors. In support of this theory is the observation that blood coming from an involved extremity has a higher eosinophil count than blood coming from the opposite extremity.[7] This suggestion has not been investigated in patients with tumors who exhibit eosinophilia.

Pisa,[8] in 1931, stated that a familial eosinophilic predisposition is necessary to provoke an eosinophilic response to malignant tumors, the foreign protein of the tumor cells being the provocative agent. In 1926 Weiss[9] had tried without success to produce this experimentally by injecting extracts of tumor tissue and blood from a patient who showed a hypereosinophilia into guinea pigs, rabbits and dogs. It is possible that the neoplastic cells must be growing to produce toxic effects and that extracts are not effective.

Isaacson and Rapaport,[1] in 1946, collected from the literature 19 cases of carcinoma showing a relative eosinophilia and concluded that the eosinophilia did not depend on the type of tumor involved, for it may occur in malignant tumors both of epithelial and of connective-tissue origin. In 29 of 30 cases, the authors demonstrated that metastases were present, and hence they concluded that eosinophilia was indicative of dissemination. They studied primary tumors thoroughly in 10 eosinophilic patients and found necrosis of tumor tissue in 6.

Dobriner and his co-workers[10, 11] first reported in 1950 that there was probably an abnormal adrenal function or altered metabolism of adrenal hormones when neoplastic disease was present. Querido,[12] in 1949, first suggested that the eosinophil count could be used in diagnosis of neoplasms.

Eosinophilia and pulmonary lesions are commonly noted to be associated, as in bronchial asthma, Loeffler's syndrome,[13-16] tropical eosinophilia, certain intestinal infestations, periarteritis nodosa, eosinophilic leukemia,[17] trichinosis and nematodes of cutaneous helminthiasis.[18, 19] Eosinophilia after lung involvement has been observed in liver abscess and hydatid cyst.[7]

Material and Methods

Patients with pulmonary lesions and white-cell counts of over 10,000 were selected for this study. Later only those with white-cell counts of over 15,000 were studied. In all, a total of 25 patients were investigated.

The propylene glycol method of Randolph,[20] as suggested by Thorn[21-23] and modified by Stephens,[24] was used as a measure of the absolute eosinophil count. Eosinophil counts were done every two hours for twenty-four hours on the first 13 patients and repeated for another twenty-four hours with the patient receiving 100 mg. of Pentobarbital every six hours. Diurnal rhythmic variations, as observed by Halberg and his associates,[25] were confirmed. The last 12 patients had two counts (three hours and one hour before the normal awakening time in the hospital) on each of three successive mornings with and without sedation.

Patients being given streptomycin treatment and those with allergic conditions were not studied because these conditions have frequently been associated with an abnormal eosinophilia.[26] Three patients studied two days before operation showed an eosinopenia. This was attributed to emotional stress.

In addition to these 25 patients, all cases of carcinoma of the lung from the preceding fifteen years at the University of Minnesota Hospital were reviewed to determine the frequency of leukocytosis in the disease.

Presentation of Data

Of the 25 patients studied, 18 had pulmonary neoplasms, 6 had lung abscess, and 1 had a lung cyst (Table 1).

*Intern, Mary Hitchcock Memorial Hospital.

It would be of value to determine whether eosinophilia is found at every level of leukocytosis when associated with neoplasm or whether it is significant only in the more severe leukocytic reactions. From this experience it appears that the absolute eosinophil count would be of the greatest value when the leukocyte count is in the range of 30,000 or above. There are too few cases here to be of significance in determining the level at which the eosinophils respond, but if in these 18 cases of carcinoma of the lung one plots the leukocyte count against the eosinophil count, one observes that the average curve points to a sharp rise at about 25,000 to 30,000 (Fig. 1). Bacterial cultures were made from the

TABLE 1. *White-Cell and Eosinophil Counts in Patients with Carcinoma, Abscess and Cyst of the Lung.*

CASE NO.	WHITE-CELL COUNT per cu. mm.	ABSOLUTE EOSINOPHIL COUNT* per cu. mm.
Patients with carcinoma:		
1	42,000	940
2	39,800	820
3	49,000	1260
4	32,000	680
5	34,000	1020
6	26,100	340
7	28,250	620
8	28,400	436
9	23,000	240
10	21,200	196
11	21,600	320
12	14,550	66
13	17,100	170
14	12,300	132
15	15,950	264
16	13,050	186
17	10,850	174
18	11,850	244
Patients with abscess:		
19	56,000	244
20	33,500	196
21	22,000	184
22	33,250	266
23	28,500	218
24	21,000	144
Patient with cyst:		
25	6,000	186

*Obtained under basal conditions.

lesion, and no evidence of pulmonary infection was found in the cases with a leukocyte count above 30,000. In 2 cases tumor and infection were accompanied by white-cell counts ranging from 20,000 to 26,000. In these cases the pulmonary process appeared on the roentgenogram as a diffuse consolidation instead of a sharply circumscribed mass, as in the 5 cases with leukocytic counts above 30,000; in all these cases there was eosinophilia (Table 1). The definite fluid level or a suggestion of one was attributed to aseptic necrosis.

In view of these observations, an attempt was made to compare the findings in the 18 cases of carcinoma with the 6 cases of nonmalignant abscess of the lung. The patients with abscess had leukocyte counts varying from 21,000 to 56,000. "Round the clock" eosinophil counts were done on these patients as described above, and not one showed an eosinophil count above 350. In the course of the investigation a case of lung cyst was found and included; this also failed to show any leukocytosis or eosinophilia.

The necrotizing cancer of the lung is a neoplasm that is apt to produce a leukocytosis, making the

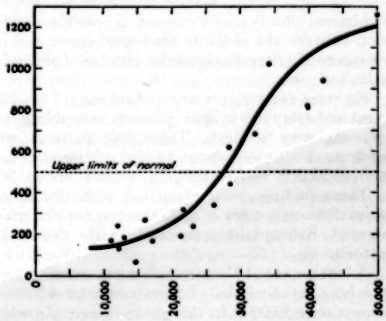

FIGURE 1. *Correlation between Absolute Eosinophil Counts (under Basal Conditions) and Leukocyte Counts in 18 Cases of Carcinoma of the Lung.*

The average preoperative leukocyte count is plotted against the absolute eosinophil count.

differentiation from an infectious process difficult. Cancer associated with a leukocytosis occurs frequently enough to present a diagnostic problem. In 122 patients with pulmonary neoplasms, the

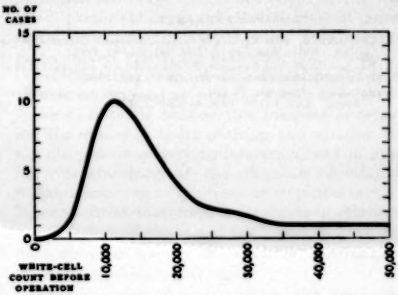

FIGURE 2. *Average White-Cell Count.*

The area under the curve represents 122 cases of carcinoma of the lung plotted according to average daily preoperative white-cell count.

incidence and degree of leukocytosis was noted (Fig. 2). From the curve it can be seen that 12.3 per cent of these patients had leukocyte counts greater than 20,000, 3.3 per cent counts greater

than 30,000, and 1.6 per cent counts greater than 40,000 cells. In patients with counts over 30,000 the absolute eosinophil count may be of help in establishing the diagnosis.

Summary and Conclusions

Limited observations suggest a possible correlation between the absolute eosinophil count and the existence of a necrotizing tumor capable of producing a leukocytosis.

Eighteen cases of carcinoma of the lung, 5 of which produced roentgenographic pictures resembling lung abscess, were studied. These five patients with necrotic tumors were found to have white-cell counts above 30,000 and eosinophil counts above 500.

These findings were compared with similar observations on 6 cases of lung abscess, none of which showed eosinophilia associated with the leukocytosis.

A review of 122 patients with carcinoma of the lung revealed that 3.3 per cent had white-cell counts over 30,000. In this group of cases, in which differentiation of a malignant from an infectious process is difficult, the eosinophil count may be a valuable test.

This investigation suggests that the toxic effects of a necrotic neoplasm produce a leukocytosis composed of all the white cells. The absolute eosinophil count could be used in the differentiation of carcinoma from infection in these cases.

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MEDICAL PROGRESS

SPATIAL VECTORCARDIOGRAPHY (Concluded)*
Review and Critique

L. Wolff, M.D.,† J. L. Richman, M.D.,‡ and A. M. Soffe, M.D.‡

BOSTON

Myocardial Infarction

The vectors of depolarization point from endocardium to epicardium in reference systems having a positive polarity, and in the reverse direction if the polarity is negative. The discussion in this and the following sections refers to systems of positive polarity, as used in this laboratory, but is applicable to the opposite polarity by a reversal of the direction of the vectors.

Each point on the QRS loop is the terminus of a resultant of numerous component vectors, contributed by all muscle elements undergoing depolarization at that instant. Infarction of some of these elements renders them electrically inert, and a new balance of forces thus ensues, so that the resultant vectors are directed more or less opposite to the infarcted areas. According to Scherlis and Grishman,[50] infarction affects the entire loop, which assumes a new spatial position diametrically opposite to the involved area. Duchosal and Sulzer[18] and Milnor[51] state that the changes are limited to the initial vectors. Observations in this laboratory indicate that the initial vectors (those occurring in the first few thousandths of a second) are altered only when the septum is grossly infarcted, in which event they point away from their normal rightward, anterior and superior position. The subsequent, but still early, vectors are altered if infarction of the free wall of the left ventricle exists; they depart from their normal disposition, and point away from the lesion. Thus, the initial or early vectors or both are those recognizably altered by infarction; changes in the entire loop are less easily evaluated. The extent of infarction, the thickness of the wall at the lesion site and heart position probably influence the magnitude and duration of the changes observed.

Duchosal and Sulzer[18] mention anterior and posterior infarcts but give no specific criteria for their localization. They state that infarction can generally be recognized by the slow inscription and abnormal direction of the initial vectors, and by a reversal in the direction of inscription. They make the important observation that the T loops are large and elongated in acute, and small and round in old infarction. Grishman and Scherlis[44, 50] limit their remarks to old lesions, and describe the criteria for the diagnosis of diaphragmatic, posterior, anteroseptal, anterior and anterolateral and combined anterolateral and diaphragmatic infarcts. By means of the direction of the initial vectors in the sagittal projection, Milnor et al.[51] attempt to differentiate normal Q deflections in Lead 3 from those indicating diaphragmatic infarction. The normal initial vectors, in the positive polarity system they use, are restricted to the −62° to +5° segment, whereas the abnormal initial vectors are distributed in the −68° to −97° segment. They also report abnormal initial vectors, indistinguishable from those occurring in diaphragmatic infarction, in cases of chronic cor pulmonale.

Myocardial infarction has been studied in this laboratory, and the results will be reported elsewhere. Briefly, we observed that open QRS loops occur early, the junction of the QRS and T loops being oriented toward the infarcted area — that is, in a direction opposite to the abnormal initial QRS vectors. Large elongated T loops, which point away from the area of infarction, then make their appearance. Finally, the changes in the initial or early QRS vectors or both mentioned above occur. Much useful information has been gathered, especially in relation to the diagnosis or exclusion of anteroseptal infarction when QS deflections occur in the right-sided precordial leads, the mimicking of anteroseptal or anterior infarction when only diaphragmatic infarct exists, the differentiation of diaphragmatic infarction from vertical heart positions and chronic cor pulmonale, and the extent of infarction.

We have made considerable progress in separating the effects of heart position and rotation from the changes secondary to infarction, and in applying this knowledge to the diagnosis of infarction without resorting to arbitrary or empirical criteria.[42] It seems to us that empirical criteria in relation to the direction of the initial QRS vectors may be as misleading as similar methods of differentiating abnormal and normal Q waves.

The diagnosis of infarction in the presence of bundle-branch block and anomalous atrioventricular excitation is discussed below.

Hypertrophy of the Right Ventricle

The literature on hypertrophy of the right ventricle is scanty and confusing. This confusion can be attributed to four factors: failure to recognize incomplete right-bundle-branch block, lack of appreciation of the magnitude of the changes pro-

*From the Electrocardiographic Laboratory, Beth Israel Hospital, and the Department of Medicine, Harvard Medical School.
Supported by a grant from the Massachusetts Heart Association.
†Clinical associate in medicine, Harvard Medical School; visiting physician and chief, electrocardiographic laboratory, Beth Israel Hospital.
‡Research fellow in cardiology, Beth Israel Hospital; post-doctorate fellow, United States Public Health Service, National Heart Institute.

duced by heart position and rotation, inadequate definition of the normal vectorcardiogram, and lack of appreciation of the effect of complicating factors such as left ventricular hypertrophy. The limitations of space do not permit an elaborate discussion of these points.

Lasser et al.[52, 53] described the vectorcardiograms from patients with congenital heart disease and lesions known to produce right-sided enlargement and classified the several patterns observed in these cases as the specific patterns of hypertrophy of the right ventricle. The most common of these patterns is characterized by clockwise inscription of the horizontal QRS loop, and disposition of the terminal QRS vectors to the right, anteriorly and superiorly. According to our concept, however, these vectorcardiograms are examples of incomplete right-bundle-branch block modified in a characteristic manner by right-chamber hypertrophy. We consider the orientation of the terminal vectors characteristic of this conduction defect, and the clockwise inscription of the whole or terminal part of the horizontal loop a modification dependent on the right-sided enlargement. Abildskov et al.[54] noted a reversal in direction of inscription of the sagittal loop in 2 of 3 cases of right ventricular hypertrophy, and we have observed similar changes. Duchosal and Sulzer[18] obtained vectorcardiograms in 2 cases of mitral stenosis and in 1 of chronic cor pulmonale. They concluded that the pattern observed in the cases of rheumatic heart disease was the result of an extremely vertical heart position, but made no comment regarding the third case. On the basis of the diagnostic criteria adopted in this laboratory one of the former is regarded as a normal vectorcardiogram, and the other 2 as examples of incomplete right-bundle-branch block modified by right-chamber hypertrophy.

According to Grishman and Scherlis[44] the T loops assume a position opposite to the QRS loop as a whole. We have been impressed with the opposition of the T vectors to the final QRS vectors, rather than to the whole QRS loop, and have noted that the T loop and the final QRS vectors are inscribed in the same direction, the tracing being similar in this respect to that in uncomplicated right-bundle-branch block.

We encountered several patterns in our cases of congenital and acquired heart disease with isolated hypertrophy of the right ventricle. Most of the vectorcardiograms were abnormal, some were bizarre, and a few were normal. The majority had features similar to those observed by Lasser et al. and by Duchosal and Sulzer. Although the observed facts are in agreement, there is, as indicated above, considerable difference of opinion regarding their explanation. Evidence obtained in this laboratory[42, 55, 56] suggests the following mechanisms: vertical heart position and clockwise rotation about the longitudinal axis of the heart, often extreme; delayed depolarization of the right ventricle; and increased right ventricular potentials. The delayed depolarization of the right ventricle is probably due to incomplete right-bundle-branch block, but another possible explanation is diffuse conduction delay peripheral to the right bundle branch.

The vertical heart position and clockwise rotation have a characteristic effect on the electrocardiogram. The initial positive deflection is small or absent in the right-sided precordial leads, which therefore display rsR, slurred, or notched R, or qR deflections. Simultaneously recorded leads, and vectorcardiograms demonstrate that the qR pattern, which is considered diagnostic of right ventricular hypertrophy,[49] occurs because the forces that ordinarily produce an r deflection in the right-sided leads are perpendicular to the lead axes and therefore have no representation in these leads.[52] In other words, instead of the small initial r wave, there is an isoelectric interval. Under these circumstances the features of incomplete right-bundle-branch block are concealed in the electrocardiogram. However, the small initial r, or slurred R deflection in electrocardiograms displaying the features of incomplete right-bundle-branch block, is strongly suggestive of right ventricular hypertrophy. The more characteristic but less common vectorcardiographic feature of right ventricular hypertrophy, the change in direction of inscription of the horizontal and sagittal projections of the QRS loop, has no counterpart in the electrocardiogram.[55, 56]

Hypertrophy of the Left Ventricle

Duchosal and Sulzer[18] offer a twofold explanation of the vectorcardiographic pattern of left ventricular hypertrophy, positional and intrinsic. They characterize the former as a horizontal heart position, and the latter as an increased magnitude of the QRS vectors and an exaggerated divergence of the T from the QRS loop. They also point out that the largest QRS vectors occur 0.045 to 0.05 second after the onset of the QRS loop, in comparison to the normal interval of 0.03 to 0.045 second. Others have noted the change in position and relation of the T to the QRS vectors[14, 57] and the QRS loop has been described as smooth and narrow,[14] as occupying a spatial position more to the left, posterior and superior, and as displaying no evidence of localized delay of inscription.[57]

Observations in this laboratory indicate that the most constant change in hypertrophy of the left ventricle is counterclockwise rotation around the longitudinal axis of the heart.[58] This occurs not only in horizontally placed hearts, the most common position in the presence of the hypertrophy, but with all other heart positions as well, including the vertical. The last is especially common in children. Counterclockwise rotation in a vertically placed heart is noteworthy, and strongly suggestive of left ventricular hypertrophy. Counterclockwise rota-

tion around the longitudinal axis of the heart produces characteristic changes in the initial QRS vectors; they have an exaggerated disposition to the right and anteriorly, with a greatly reduced or minimal upward direction.[42] The intrinsic changes — that is, the increased magnitude of the QRS vectors and the altered relation between T and QRS loops — have been observed in varying degrees, or may be absent. In some cases there is pronounced slowness of inscription of the terminal part of the QRS loop, but this is the least common feature of the left ventricular hypertrophy. It is not known whether these changes are due to increased thickness of the ventricular musculature, to focal intraventricular block or to some other condition.

The direction of inscription of the T loops is the same as that for the QRS loops.[55]

The characteristic changes of left ventricular hypertrophy just described are clearly evident in vectorcardiograms displaying the diagnostic signs of right-bundle-branch block.[54, 57] We have not been able to confirm the observations of other investigators, who have found diagnostic signs of left ventricular hypertrophy in the presence of left-bundle-branch block.[59]

Combined Hypertrophy of Right and Left Ventricles

The vectorcardiographic patterns of combined ventricular hypertrophy have not been described adequately. Mann[9] reported 2 cases, but only the frontal-plane projections of the spatial vectors were available to him.

The following is a summary of our observations, which will be reported in detail elsewhere. Combined hypertrophy alters the electric field surrounding the heart in several ways, and produces three general types of vectorcardiogram.[55, 56]

In congenital heart disease the vectorcardiogram resembles that described above in cases of isolated hypertrophy of the right ventricle and differs from it in that there is counterclockwise, instead of clockwise, rotation around the longitudinal axis of the heart. The reverse rotation is revealed by the exaggerated rightward and anterior and minimal superior orientation of the initial QRS vectors, and the counterclockwise inscription of the frontal QRS loop. The T vectors and the final QRS vectors point in opposite directions.

In acquired heart disease (rheumatic heart disease and 1 case of essential pulmonary hypertension) the vectorcardiogram differs from that seen in isolated right ventricular hypertrophy in that the terminal QRS forces are directed posteriorly and less superiorly, while retaining the other features of right ventricular hypertrophy. The T and final QRS vectors point in opposite directions.

In a second group with acquired heart disease (rheumatic heart disease or chronic cor pulmonale) the vectorcardiograms display marked clockwise rotation with terminal appendages oriented posteriorly, superiorly and to the right. The T loops are abnormal, but exhibit no constant pattern.

There are many reports on the electrocardiographic features of combined ventricular hypertrophy, but no agreement whether the diagnosis of combined chamber enlargement can be made and, if so, on what basis.[49, 60-68] This doubtful status of the electrocardiogram is in striking contrast to the evidence of combined ventricular hypertrophy revealed by the vectorcardiogram, and is dependent on the fact that the direction of inscription of the QRS loop, which is important for diagnosis, is not revealed by scalar derivations of the cardiac vector. We have re-evaluated the electrocardiograms in our cases of combined ventricular hypertrophy studied by vectorcardiogram, and have observed the following patterns: in the cases of congenital heart disease the features are those of right-bundle-branch block and marked counter-clockwise rotation; in acquired heart disease the tracings are similar to those seen in isolated right ventricular hypertrophy but late negative deflections (S or S′) occur in the right-sided precordial leads, or the tracings cannot be distinguished readily from normal electrocardiograms, except that the T waves are abnormal.[55, 56]

Right-Bundle-Branch Block

The QRSsÊ loop of right-bundle-branch block can be divided theoretically into two parts. The first represents depolarization of the interventricular septum from left to right, and the free wall of the left ventricle. The second corresponds to depolarization of the free wall of the right ventricle. Duchosal and Sulzer[16] state that the second part of the loop represents depolarization of the inferior part of the septum as well as the right ventricular wall. Lasser and Grishman[54] observed, in cases of intermittent right-bundle-branch block, that the initial vectors are somewhat more anterior during defective conduction, but in common with Abildskov et al.[54] and Duchosal and Sulzer,[16] we found no appreciable difference in such cases. Evidently, the normal right ventricular potentials are small — a fourth the magnitude of those of the left ventricle, according to Duchosal. The late QRS vectors are directed anteriorly, superiorly and to the right, and the terminal part of the QRS loop is inscribed slowly and irregularly in a counterclockwise direction in the horizontal and in a clockwise direction in the sagittal projections.

The first part of the QRS loop, therefore, reveals the status of the septum and left ventricular myo-

cardium, and the second part of the loop that of the right ventricular muscle.[64, 66, 68] Like Abildskov et al.[64] we recognize a variety of right-bundle-branch block that is characteristic of right ventricular hypertrophy as discussed above. A contrary view is expressed by Lasser and Grishman,[64] who are of the opinion that there are no special features in right-bundle-branch block that are diagnostic of right ventricular hypertrophy.

There is also considerable difference of opinion concerning the relation of the QRS and T vectors in right-bundle-branch block. Duchosal and Sulzer[16] and Vastesaeger[66] believe that the T loop, like the QRS loop, can be divided into two parts, the first corresponding to septal and left ventricular repolarization, and the second to right ventricular and inferior septal repolarization. Conway and his associates[14] found the T vectors in right-bundle-branch block to be the same as those in the presence of normal intraventricular conduction. The findings in this laboratory are in agreement with those of Grishman and Scherlis,[64] who noted that the T vectors are opposite to the late QRS vectors in the absence of myocardial disease. We have made the additional observation that the T and late QRS vectors are inscribed in the same direction — that is, counterclockwise in the horizontal, and clockwise in the sagittal planes.

Scherlis et al.[41] and Lasser and his co-workers[67] distinguish incomplete from complete block by virtue of the shorter duration of the slowly inscribed terminal portion of the QRS loop, which is contrary to our observations that localized slowness of inscription may be entirely absent in the lesser grades of this conduction defect. Furthermore, we have noted that the terminal QRS vectors in incomplete right-bundle-branch block, though characteristically directed to the right and superiorly, are not necessarily oriented anteriorly. In other words, the vectorcardiograms that Lasser et al.[67] described as normal but with a terminal appendage are, we believe, examples of incomplete right-bundle-branch block.

Left-Bundle-Branch Block

There is lack of agreement concerning the vectorcardiographic features of left-bundle-branch block. Scherlis and Grishman[67] restrict the diagnosis to vectorcardiograms having the following characteristics: the initial QRS vectors are directed posteriorly, superiorly and to the left; the QRS$_{3}$Ê loop is oriented posteriorly, superiorly and to the left; the horizontal projection is inscribed in a clockwise direction or has a figure-of-eight configuration; and there is delay of inscription in the middle of the QRS$_{3}$Ê loop. Sodi-Pallares[68] considers some of the cases that Grishman and Scherlis[64] diagnosed as hypertrophy of the left ventricle to be examples of left-bundle-branch block. Conway et al.[14] do not recognize a characteristic position of the QRS$_{3}$Ê loop, but base the diagnosis on early, "sudden" middle and late delayed inscription of the vectors. Duchosal and Sulzer[16] briefly comment that the QRS vectors enlarge in the sagittal and transverse axes, but do not mention slow inscription. Observations in this laboratory[69] indicate that the only invariable finding in complete and incomplete left-bundle-branch block is the direction of the initial vectors, which point to the left, anteriorly and inferiorly. The small initial vectors are responsible for the heavily slurred R deflection in the left-sided precordial leads, which Sodi-Pallares and his co-workers[70] consider characteristic of left-bundle-branch block. The general disposition of the loops varies considerably; the horizontal loop may be inscribed in a clockwise or counterclockwise direction, or may have a figure-of-eight configuration, and there may or may not be localized delay of inscription.[69]

The initial depolarization process in left-bundle-branch block involves two muscle masses, the interventricular septum, which is depolarized in a right-to-left direction, and the free wall of the right ventricle. The powerful forces of the left ventricle are absent from the early phase of ventricular activation, since the depolarization process reaches the left side of the heart belatedly. Therefore, unlike the situation existing in right-bundle-branch block, the entire loop, including the initial QRS vectors, is affected. Scherlis et al[67] preclude the possibility of recognizing evidence of myocardial disease because of the wholly abnormal order of ventricular excitation. Pantridge and his co-workers,[59] on the other hand, have interpreted some of the patterns diagnosed by them as left-bundle-branch block as giving evidence of the underlying status of the myocardium, but they do not present adequate support of their contention. We believe the solution to this problem may be found in the fact that the early QRS vectors represent septal and right ventricular and the late forces left ventricular and perhaps septal activation. Therefore, infarction of the septum alters the early QRS vectors, and infarction of the free wall of the left ventricle the late QRS vectors.[71]

The T loop is opposite to the QRS loop,[14] and is inscribed in the same direction as the QRS loop.[69]

Intraventricular Block

The vectorcardiogram is a fruitful method of studying intraventricular block, but practically nothing has appeared in the literature to date. We have observed two types of intraventricular block, one in cases with electrocardiograms displaying the diagnostic signs of arborization block, and the other a type not heretofore described.[72, 73]

The vectorcardiograms in cases of arborization block display signs of myocardial infarction. The terminal portion of the QRS loop is inscribed slowly,

and the vectors point in a diametrically opposite direction to the early abnormal vectors.

The previously undescribed type of intraventricular block has some features of left-bundle-branch block but differs from it in other respects. The following features are characteristic: the initial vectors are directed anteriorly, inferiorly and to the left, in most cases; the terminal QRS vectors point to the right and posteriorly, and markedly superiorly; the QRS loop is large and is directed essentially superiorly; the duration of the QRS loop is prolonged in most but not in all cases; the terminal part of the QRS loop is inscribed slowly; the horizontal and frontal loops are inscribed in a counterclockwise and the sagittal loop in a clockwise direction; and the T loops point in an opposite direction to the QRS loops, and are inscribed in the same direction.

The electrocardiogram displays marked left-axis deviation, Q waves in Leads 1 and aVL, tall late R waves in Lead aVR, and RS deflections in Lead aVF and the left-sided precordial leads. The QRS interval is normal, or is prolonged — at times markedly so.

This abnormality probably represents block in a specific area of the specialized conduction system on the left side of the heart.[72]

Short PR Interval and Abnormal QRS Complexes (Wolff–Parkinson–White Syndrome) (Anomalous Atrioventricular Excitation)

The most striking feature is the unusually slow inscription of the initial segment of the QRS loop.[16, 44, 74, 75] This segment, as a rule, is straight and directed to the left, without significant anterior or posterior deviation; it may point upward or downward, and is of variable duration. It is evident, since the anomalous segment can be identified in the QRS loop, that measurement of atrioventricular conduction time is not essential for the vectorcardiographic diagnosis of the syndrome. Indeed, it has been recognized in the presence of persistent auricular fibrillation in 1 of our cases.

Segers et al.[74] are of the opinion that the subsequent vectors are normal, but Grishman and Scherlis[44] disagree with this view. Duchosal and Sulzer[16] state that the late vectors are abnormal only when the "anomalous component" is premature by more than 0.08 second. Some of the cases studied in this laboratory displayed anomalous conduction intermittently, and served to demonstrate that the vectors following the anomalous segment may be normal. Moreover, when the end of the anomalous segment can be identified, it is possible to diagnose myocardial infarction[75] and hypertrophy of the right ventricle in the presence of anomalous conduction, contrary to the opinion of Grishman and Scherlis.[44] Right-bundle-branch block and anomalous conduction may exist together. Both mechanisms are displayed simultaneously by the vectorcardiogram.

Grishman and Scherlis[44] state that the T loop is more or less opposite to the QRS loop, but we have found no constant relation in our cases.

The vectorcardiogram is superior to the electrocardiogram in the study of anomalous atrioventricular excitation in three respects: the diagnosis can be made in the presence of auricular fibrillation; myocardial infarction and hypertrophy of the right ventricle may be recognized without the necessity of changing the cardiac mechanism; right-bundle-branch block can be identified during anomalous atrioventricular conduction.[75]

Miscellaneous Anomalies

Vectorcardiographic studies of many other abnormal states, such as electrolyte imbalance, pulmonary embolism, myocarditis and the effect of drugs, have not yet appeared in the literature.

The Place of Vectorcardiography in Medicine

An estimate of the place that vectorcardiography may rightfully occupy in medicine requires that it be compared with electrocardiography, since the immediate and ultimate aims of both methods of examination are the same. An evaluation of this sort must take into account the comparative difficulties in technic and interpretation, the comparative costs and, most important of all, the accuracy and usefulness of the data obtained by each procedure. Above all, the possibility must be considered that the new method may extend the frontiers of knowledge of the cardiac electromotive force beyond the limits apparently fixed by electrocardiography.

Experience with vectorcardiography has convinced us that the technic is much more difficult than that required for obtaining electrocardiograms. This is especially so when certain reference systems are used, making it obligatory to identify anatomic landmarks with the utmost care, and to revise the standardization from lead to lead. As a rule, detailed study of the P, QRS and T loops necessitates the employment of several photographs, each with appropriate standardization, obtained at split-second intervals. Imperfections of registration commonly occur because of restlessness, hyperventilation, rhythmic contraction of some muscle mass or somatic tremor, and it may be impossible to obtain vectorcardiograms in infants and children who are struggling or crying. Haziness or drifting of the luminous spot or small shifts in the trace are common, and may result from any of these factors or from other extraneous influences. These produce artefacts in the P, T and QRS loops, and may lead to wholly erroneous interpretation, or they may obscure the initial vectors sufficiently to render the tracings useless. The constant electric activity of the auricles in fibrillation or flutter similarly may interfere with the recording of a distinct trace, especially the beginning of the QRS loop. The

importance of achieving clear definition of the initial QRS vectors is discussed above.

The cost of obtaining vectorcardiograms must be measured in terms of time and money. It takes four or five times longer to obtain suitable vectorcardiograms than electrocardiograms with a full set of leads (12 or 13 leads), and preparation of the vectorcardiograms for study takes a good deal of time as compared to the immediate availability of electrocardiograms. When emergency or frequent and repeated tracings are required, all difficulties are multiplied accordingly. Furthermore, the difficulties involved in constructing a portable outfit limit the application of the method to hospital patients who can be moved, and to ambulatory persons. And, finally, the instruments and materials required for vectorcardiography are expensive. The total cost of oscilloscope, special amplifiers, tuning fork and camera amounts to $2,500 to $5,500, compared to $500 to $700 for a good commercial electrocardiograph. The film used in vectorcardiography is considerably more expensive than electrocardiographic paper, and the cost of developing and printing is also greater.

Interpretation is undoubtedly the most difficult aspect of vectorcardiography. Principles of interpretation have not, as yet, been formulated and accepted generally. Lack of agreement among different investigators concerning methods stands in the way, and probably will continue to do so for a long time. In this laboratory, where simple rules have been adopted, the average time required for interpretation of a single record is approximately fifteen minutes.

These difficulties render the vectorcardiographic method unsuitable for routine use at present.

An appraisal of the intrinsic value of vectorcardiography will undoubtedly lead to different opinions among various investigators. The limitations of the method have been commented upon,[76] and attitudes have varied from cautious restraint[77] to the prediction that this is the method of the future.[78] The final answer must await the concerted effort of many investigators, over a considerable period, and it is to be hoped that the direct experimental approach, which did so much to establish electrocardiography as a fundamental method of investigation, will be utilized to the fullest extent. Nevertheless, some remarks of a factual nature are warranted.

We have already pointed out that arrhythmias cannot be studied if still photographs are employed, but this deficiency can be remedied by the use of motion pictures. The PR and QT intervals cannot be measured, but the P, QRS and T intervals are measurable.

The vectorcardiogram, unlike the electrocardiogram, gives the spatial position of the cardiac vectors. Therefore, the electric position of the heart is ascertainable in greater detail in vectorcardiograms, and can be determined in many cases even when there is localized myocardial disease. The effect of heart position and rotation on the cardiac vector can be studied experimentally by vectorcardiographic methods, and the distinction between the extrinsic effects of position and the intrinsic effects of myocardial changes can be clearly defined.[42]

Vectorcardiography has focused attention on many fundamental questions, among them the significance and position on the QRS loop of the intrinsicoid deflection, the validity of unipolar precordial leads, the genesis of various normal and abnormal patterns and the spatial position of the ventricular gradient. Furthermore, the direction and speed of inscription of successive resultant vectors during activation and recovery, and the spatial relation between the QRS and T loops is evident in the vectorcardiogram, but not in the electrocardiogram.

From a diagnostic point of view we have come to the following conclusions, based on experience gained from a study of more than 800 patients. The vectorcardiogram is superior to the electrocardiogram in the diagnosis, localization and estimation of extent of myocardial infarction, and permits the recognition of infarcts so situated that diagnostic signs are not produced in scalar derivations. The diagnosis of infarction in the presence of left-bundle-branch block and anomalous atrioventricular excitation is possible with the new method of examination. It cannot be stated as yet how often, or under what circumstances, myocardial infarction exists without producing reliable diagnostic signs in the vectorcardiogram, and it is not known how often such diagnostic signs are present in the absence of a myocardial lesion. The vectorcardiogram is superior to the electrocardiogram in the recognition of incomplete left-bundle-branch block, and in the diagnosis of right ventricular hypertrophy and combined ventricular hypertrophy. Its superiority in the diagnosis of right ventricular hypertrophy and in clarifying the mechanism of the changes in this condition is especially noteworthy. The diagnostic limitations in relation to ventricular hypertrophy are similar to those just expressed for myocardial infarction.

Summary

Vectorcardiography is a useful method of investigation of the electric phenomena associated with the heart beat. It has already extended basic knowledge in this field, and there is every indication that this progress will continue. From a diagnostic point of view the evidence is clear that vectorcardiography is superior to electrocardiography, with the exceptions noted above. Although the method is not yet simple enough, or well enough established, for widespread adoption, it has already produced benefits out of proportion to its limited

use by clarifying many equivocal electrocardiographic signs. Vectorcardiography, therefore, should be developed and extended, despite the present technical difficulties.

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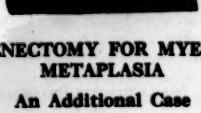
MEDICAL INTELLIGENCE



SPLENECTOMY FOR MYELOID METAPLASIA

An Additional Case

BERT SELIGMAN, M.D.*

TOLEDO, OHIO

IN view of the recent report in the *Journal of* 5 cases of myeloid metaplasia treated by splenectomy,† the following case may be of interest.

Case Report

Mrs. I. M., a 64-year-old housewife, was admitted to Flower Hospital, Toledo, Ohio, on January 20, 1950. She had noted progressive enlargement of the abdomen during the past year, with epigastric fullness, distress after a heavy meal and pain and bleeding from hemorrhoids.

Physical examination revealed a large, hard, nontender mass in the left upper quadrant of the abdomen, which appeared to be an enormous spleen.

The red-cell count was 3,840,000, with a hemoglobin of 12 gm. per 100 cc., and the white-cell count 3950, with 82 per cent segmented cells and 18 per cent lymphocytes. The sedimentation rate was 5 mm. in 60 minutes. The total protein was 5.94 gm. per 100 cc., with an albumin of 3.41 and a globulin of 2.53 gm.

A diagnosis of agnogenic myeloid metaplasia was made, and on January 24, splenectomy was carried out.

The spleen measured 25 x 18 x 9 cm. and weighed 1690 gm. Sections revealed great numbers of immature myeloid cells and megakaryocytes. Nucleated red cells were also numerous. Liver biopsy showed numerous megakaryocytes squeezing through sinuses, and small foci of round cells about the portal fields.

After splenectomy on June 13, the red-cell count was 5,400,000, with a hemoglobin of 102 per cent, and the white-cell count 26,900, with 9 per cent stab and 81 per cent segmental forms, 5 per cent lymphocytes, 3 per cent monocytes and 2 per cent eosinophils. There were 3 nucleated red cells, and the hematocrit was 64 per cent. The blood urea nitrogen was 11.5 mg., and the calcium 12 mg. per 100 cc., and the chloride 120 milliequiv. per liter. The total protein was 6.64 gm. per 100 cc., with an albumin of 2.99 and a globulin of 3.65 gm.

On June 10 the patient was seen in congestive heart failure. An electrocardiogram 2 days later was interpreted as showing first-degree auriculoventricular block, preponderance of the left ventricle and myocardial changes with the possibility of an anterior infarct. She died on June 16. Autopsy demonstrated status postsplenectomy, cardiac hypertrophy, arteriolar sclerosis of the kidneys, pitting edema, suppurating bronchitis, bronchiolitis and bronchopneumonia, pulmonary atelectasis, abdominal adhesions and fibrosis of the bone marrow. The report further stated that these findings were compatible with a diagnosis of agnogenic myeloid hyperplasia.

Conclusions

Removal of the spleen was not catastrophic to the patient nor to the blood-forming system. In well selected patients with severe thrombocytopenia splenectomy may be of benefit.

*Senior surgeon for vascular surgery, Flower Hospital; active member, Division of Peripheral Vascular Surgery, Maumee Valley Hospital.

†Green, T. W., Conley, C. L., Ashburn, L. L., and Peters, H. R. Splenectomy for myeloid metaplasia of spleen. *New Eng. J. Med.* 249:211-219, 1953.

QUEENS DIE BRAVELY

IN view of the present period of mourning in the British Commonwealth for the late Queen Mary, the following account* of the circumstances surrounding the death of another Queen Mary (wife of William of Orange), in 1694, is of interest.

He [William of Orange] had but too good reason to be uneasy. His wife had, during two or three days, been poorly; and on the preceding evening grave symptoms had appeared. Sir Thomas Millington, who was physician in ordinary to the king, thought that she had the measles. But Radcliffe, who, with coarse manners and little book learning, had raised himself to the first practice in London chiefly by his rare skill in diagnostics, uttered the more alarming words, small pox. That disease, over which science has since achieved a succession of glorious and beneficent victories, was then the most terrible of all the ministers of death. The havoc of the plague had been far more rapid but the plague had visited our shores only once or twice within living memory; and the small pox was always present, filling the churchyard with corpses, tormenting with constant fears all whom it had not yet stricken, leaving on those whose lives it spared the hideous traces of its power, turning the babe into a changeling at which the mother shuddered, and making the eyes and cheeks of the betrothed maiden objects of horror to the lover. Towards the end of the year 1694, this pestilence was more than usually severe. At length the infection spread to the palace, and reached the young and blooming Queen. She received the intimation of her danger with true greatness of soul. She gave orders that every lady of her bedchamber, every maid of honour, nay, every menial servant, who had not had the small pox, should instantly leave Kensington House. She locked herself up during a short time in her closet, burned some papers, arranged others, and then calmly awaited her fate.

During two or three days there were many alternations of hope and fear. The physicians contradicted each other and themselves in a way which sufficiently indicates the state of medical science in that age. The disease was measles: it was scarlet fever: it was spotted fever: it was erysipelas. At one moment some symptoms, which in truth showed that the case was almost hopeless, were hailed as indications of returning health. At length all doubt was over. Radcliffe's opinion proved to be right. It was plain that the Queen was sinking under small pox of the most malignant type.

. . .Tenison undertook to tell her that she was dying. He was afraid that such a communication, abruptly made, might agitate her violently, and began with much management. But she soon caught his meaning, and, with that meek womanly courage which so often puts our bravery to shame, submitted herself to the will of God. She called for a small cabinet in which her most important papers were locked up, gave orders that, as soon as she was no more, it should be delivered to the King, and then dismissed worldly cares from her mind. She received the Eucharist, and repeated her part of the office with unimpaired memory and intelligence, though in a feeble voice. She observed that Tenison had been long standing at her bedside, and, with that sweet courtesy which was habitual to her, faltered out her commands that he would sit down, and repeated them till he obeyed. After she had received the sacrament she sank rapidly, and uttered only a few broken words.

*Macaulay, T. B. *History of England.* Vol. 5. Boston: Macmillan, 1913. Pp. 2668-2670.

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Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*

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CASE 39201

PRESENTATION OF CASE

A thirty-five-year-old woman was admitted to the hospital because of pain in the left hip.

Five years previously she first began to experience this pain after activity. The pain was constant in distribution, affecting the left hip, thigh and knee and extending into the posterior calf and dorsum of the foot, and gradually increased in severity. Four and two and a half years before admission, when she was studied at another hospital, physical examination was negative, and x-ray films of the spine and hip were normal. The possibility that the symptomatology was on a psychosomatic basis was seriously considered. The pain continued to increase in severity and became especially severe during the six months before admission, and she was forced to stop her secretarial work three months before admission. The pain no longer subsided with bed rest, although she was most comfortable with the hip and knee flexed. The pain was exaggerated by coughing and sneezing. There had been no constitutional symptoms except that she tired easily when the hip was painful. The left heel occasionally became numb, but there were no other paresthesias or vasomotor symptoms. Non-weight-bearing crutch walking afforded some relief, but at the end of a week there was an acute exacerbation of the pain. No change in the symptomatology was noted after a week of cortisone therapy.

During early childhood the patient had been overweight and had worn braces because of bowlegs. Ten years before entry she had a hysterectomy, complicated postoperatively by thrombophlebitis; recovery from this was uneventful, and she maintained that the pain in the legs at that time was "entirely different" from that experienced in the present illness. Psoriasis of both legs had been present for ten years, being most severe during the winter.

Physical examination revealed psoriasis of both lower legs. There was questionable decreased motion of the spine. There were pain on palpation of the left femoral head anteriorly and posteriorly, and an area of paresthesia over the left lateral hip and thigh, popliteal space and posterior calf. Movements of the hips were as follows: extension — left 0°, right 10°; flexion — left 110°, right 110°; internal rotation — left 10 to 15°, right 35°; external rotation — left 15 to 20°, right 35°; abduction — left 15°, right 30°; and adduction — left 20°, right 20°. The reflexes were equal and active bilaterally. The remainder of the examination was negative.

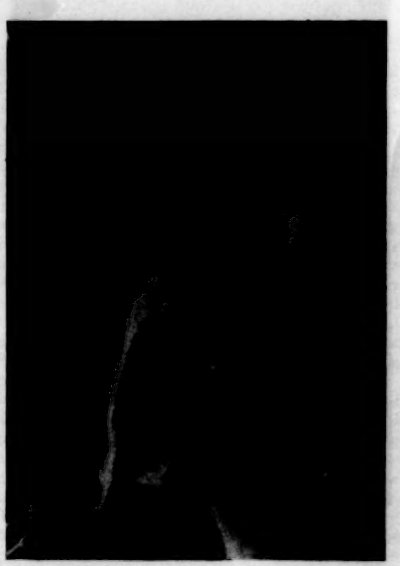

FIGURE 1. *Anteroposterior View of the Left Hip. (The Fracture Lines Seen on the Films Cannot Be Adequately Reproduced.)*

The temperature was 98.6°F., the pulse 85, and the respirations 20. The blood pressure was 130 systolic, 70 diastolic.

Examination of the blood showed a hemoglobin of 13.8 gm., a white-cell count of 10,400, with 59 per cent neutrophils, a hematocrit of 43 per cent and a corrected sedimentation rate of 0.94 mm. per minute. The urine was normal. A blood Hinton test was negative. The serum calcium was 9.8 mg., the phosphorus 4.3 mg., the alkaline phosphatase 4.3 units, the uric acid 4.0 mg., and the cholesterol 260 mg. per 100 cc. The brucella agglutination

and the Widal test were negative. A 1:10,000 tuberculin test showed a 1-cm. subcutaneous area of induration, without redness, after forty-eight hours.

Roentgenograms of the chest were normal. Anteroposterior and lateral views of both femurs gave evidence of moderate osteoporosis of the left femur and an intrinsic bony lesion on this side in the region of the femoral neck and head (Fig. 1 and 2). The lateral aspect of the femoral neck and contiguous portion of the head was largely devoid of a trabecular pattern, and the region had a smudged appearance. The femoral articular cortex was slightly irregular, and there was evidence of periosteal reaction at the junction of the head and neck, most marked anteriorly and posteriorly. At this level there were a number of transverse sclerotic linear areas. There was no growth on culcure of material aspirated from the lesion.

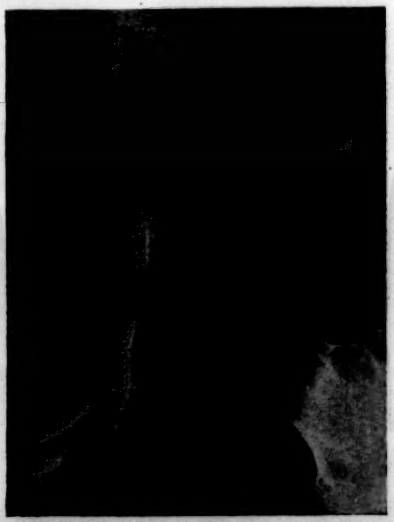

FIGURE 2. *Lateral View of the Left Hip.*

Traction applied to the left leg had no effect on the symptoms. There was an occasional temperature rise to 100°F. On the fourteenth hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. OTTO E. AUFRANC*: The data on hip motions are interesting if they are accurate. A flexion of 110° is not normal for either hip.

May I see the x-ray films?

*Assistant orthopedic surgeon, Massachusetts General Hospital.

In evaluating lesions of this type one must be able to see both hip joints for comparison. In these views (Fig. 1 and 2) there is a tendency to subluxation of the left hip. There is widening of the acetabular joint line inferiorly and superiorly and a fracture of the neck, as well as of the acetabulum, that I am sure is pathologic. There is an area of diminished trabecular pattern on the superior lateral aspect of the neck of the femur extending down to the base and up to and including part of the head. A fairly marked osteoporosis is present in most of the neck, above the acetabular joint line, in the ischium and in the ilium, and extends up to the sacroiliac joint. The pathologic fracture of the neck of the femur is seen, with buckling up of the periosteal bone at the fracture line. The trabecular pattern is increased along the margin of the acetabulum, which I believe also represents a partial fracture. I can find no evidence of soft-tissue tumor anywhere. I think it is important to see this difference in the two hips at the joint line; the involved left hip has a wider line. This is seen particularly well in the anteroposterior view. This gives me a lead, or a mislead, to the diagnosis.

I should like to discuss one or two points in the physical examination that I think are important. If the normal hip had only 110° flexion, the involved hip must have had permanent flexion, because the patient was in bed with the hips flexed or nearly flexed, which is evidence of limitation of motion in extension. The other consideration is that the symptoms were confined to the area of the hip. The onset of pain before admission was consistent with a pathologic fracture that is seen on x-ray examination. The first x-ray films taken five years before admission, which were reported as normal, require comment. In those films there is a wide bifid sacrum, which I think is important in view of the patient's pain pattern, especially if this new lesion noted in the films was not causing the pain. I think the lesion in the joint was not five years old; it seems to have been too rapidly growing to have been that old. Occasionally, I have had a patient with proved ruptured intervertebral disk who will say he has had hip pain and point to the sacrum, the sciatic nerve and down the back of the leg and so on. The patient under discussion had a congenital anomaly of the back that could have produced hip, posterolateral thigh, groin and right leg pain. I think that was the cause of the old pain.

The differential diagnosis, then, is between a tumor and an infectious lesion? I think the clinical course is against an infectious lesion; moreover, the blood count, the temperature and the general condition of the patient seem to be against that. Hence tumors either primary or secondary are the next possibilities to consider.

One question I should like to ask is, Why did this patient have a hysterectomy at the age of twenty-five?

DR. CHARLES L. SHORT: The uterus was removed for leiomyomas.

DR. AUFRANC: It was not for cancer.

The benign lesions in this area of the hip are the chondromas, — enchondromas and osteochondromas, — the synovitides and monarticular arthritis. I should like to eliminate the localized joint lesion of arthritis on the basis that there seems to have been only bone destruction in this area. The trabecular pattern was lost in the superior portion — this was particularly noticeable in the anteroposterior view; the lesion seems to have had a more or less definite outline of involvement, seems to have broken through the cortex in the posterior neck and seems also to have traversed the joint ring. A villonodular synovitis occasionally presents this picture, but I have yet to see a pathologic fracture associated with such a diagnosis. I should mention in passing that metabolic diseases, disturbances in the blood and myeloma can cause bone lesions and should be considered, but the physical findings and the laboratory work seem to point to other conditions.

A lesion that produces no soft-tissue tumor mass and seems to confine itself to the bone and joint makes me believe I have to consider mainly a metastatic lesion or a primary tumor of the bone itself. Metastatic disease could have arisen either from the breast or from some other remote source. In the physical examination there was no evidence of this. Metastatic lesions, it seems to me, can be ruled out by the evidence from the x-ray appearance and the physical findings that this tumor involved both sides of the joint. I may be entirely wrong in interpreting this film, but it seems to me that that is true.

I have brought my diagnoses down to a primary tumor of the bone in the area of the neck of the femur; the most likely diagnosis is some kind of sarcoma, a fibrosarcoma, a chondrosarcoma of an enchondral type or perhaps a myxosarcoma, any one of which could produce this picture. Another tumor that should be considered is an eosinophilic granuloma, which I believe does not occur often in this age group. I shall vote for a primary bone tumor possibly a fibrosarcoma.

DR. SHORT: I saw this patient from a medical point of view and could find no evidence of any systemic or metabolic disease to account for the findings. I thought from the symptoms that she probably had a spinal lesion, but I could not localize it or determine what its nature was. The sedimentation rate was increased, and she had a slight fever, which brought up seriously the question of an infectious process, but I agreed with Dr. Aufranc that the rest of the picture did not fit infection. She had no form of arthritis with which we are familiar.

DR. AUFRANC: One point about bone lesions that I am sure every surgeon believes is that he needs a knife, an osteotome, and a pathologist to diagnose these lesions.

CLINICAL DIAGNOSIS

?Aseptic necrosis, left femoral head.

DR. OTTO E. AUFRANC'S DIAGNOSIS

Fibrosarcoma of femur.

ANATOMICAL DIAGNOSIS

Chondrosarcoma of femur.

PATHOLOGICAL DISCUSSION

DR. JOHN D. LEIDHOLT: Dr. Joseph S. Barr and I saw this lesion at operation. As we exposed the area, the overlying soft tissue and the outer surface of the hip capsule appeared to be normal — perhaps slightly more vascular. When the joint capsule was opened it was apparent that the entire synovial membrane was thickened to about 0.6 cm. The neck of the femur was visualized, and the cortex appeared to be very thin, so thin in fact that merely by palpating it with the finger or with an elevator one could push right through it onto the superior surface. Within the medullary cavity was an almost homogeneous pale-gray granular material that contained small spicules of bone. This filled up the entire area of the neck, a good part of the head and as far distally as we could see, extending beyond the intertrochanteric line. The most striking thing to me was the marked involvement of the synovia, which was borne out when we inspected the ligamentum teres, which in addition to being 1 cm. in thickness, was shortened and extremely edematous. The fluid within the joint seemed to be increased in quantity but was normal in appearance. The fracture that Dr. Aufranc observed in the x-ray films was apparent grossly as a deformity of the proximal end of the femoral neck, indicating an old pathologic fracture.

DR. DAVID KAHN: The first specimen we received consisted of curettings from the neck of the femur for frozen section. However, even with the last third of the requisites that Dr. Aufranc mentioned, I was unable to make a definite diagnosis. The section consisted of fibrous bone marrow and cartilaginous material, with spicules of osteoid and new bone; in the face of the fracture, we were unable to say for certain whether this represented an area of fibrous dysplasia, with a fracture through it, and callus formation, or whether it was a true cartilaginous tumor. Since we could not give a definite diagnosis, since there was a chance that the lesion was benign, and since the femoral head was so distorted as to be no longer serviceable for weight bearing, Dr. Barr did a resection of the femoral head and neck and inserted a Moore prosthesis. This slide (Fig. 3) is of a cross section of the removed head and neck. The head was distorted, and the articular cartilage showed degenerative

change. The empty space in the medullary cavity of the neck was the region from which the biopsy for frozen section was taken. A definite lesion occupied almost the entire neck and a portion of the head. Here the normal pattern of spongiosa was lost and replaced by an admixture of gray, white and pearl-colored tissue. The linear gray line represented the healing fracture. The lesion extended to the articular cartilage at one point and to the

FIGURE 3.

cortex of the femoral neck, which was thinned out. Grossly, there was no break through into the surrounding tissue. As was known at the time of operation, the lesion extended to the resection margin. Also received were a thickened ligamentum teres and pieces of thickened synovia.

Histologically the lesion was a chondrosarcoma. The tumor was composed of fairly large polygonal cells with centrally placed nuclei and without much atypicality; these cells bordered on each other, and nowhere were they producing much cartilaginous ground substance. In our preliminary sections we were struck by the resemblance of the cells to those of benign chondroblastoma, a tumor of the epiphysis that occurs in adolescence. However, because tumors of this group have not been reported beyond the age of adolescence, we were a little skeptical of this diagnosis. Another outstanding feature in this tumor was the presence of small spicules of new bone. This was bone forming in cartilage, which is not uncommon in any cartilaginous tumor—chondroma, chondroblastoma or chondrosarcoma. In other sections fields were found in which the tumor cells were definitely atypical and had atypical, sometimes hyperchromatic nuclei. Some cells had double and triple nuclei. These findings in a cartilaginous tumor are definitely indicative of sarcoma. The tumor had invaded up to the articular cartilage and into the cortex of the neck, which was thinned out, but not completely through it. This was a central chondrosarcoma and we were unable to find any evidence that it had broken through its bony boundaries. In a number of sections tumor cells were found invading vascular channels, and a few small veins contained plugs of tumor tissue.

In addition to metastasizing to the lungs via tumor emboli, chondrosarcoma can grow by direct extension along veins, and this fact must be taken into consideration in the treatment of these tumors. As we expected from the gross inspection, the tumor extended to the resection margin. An interesting feature in this tumor was that with the exception of the small bony spicules, there was almost a total absence of calcification; this fact accounts for the absence of stippling usually seen in the x-ray films of cartilaginous tumors. The ligamentum teres and synovial membrane showed marked chronic synovitis, but no evidence of tumor.

We seriously wondered if this tumor represented a malignant transformation of a pre-existing benign chondroblastoma. Since no malignant counterpart of benign chondroblastoma has been reported, we showed the sections to a number of people who have wide experience with benign chondroblastoma, and at present count there is about an even division among those who think it may have been and those who think it was not related to chondroblastoma.

Dr. Taylor reoperated on this patient, performing a hemipelvectomy. As was expected residual tumor was found in the proximal few inches of the femoral shaft. No tumor was found in the soft tissue, veins, lymph nodes or other bones.

Dr. Taylor, would you like to comment?

DR. GRANTLEY W. TAYLOR: We were confronted with a difficult problem. I saw this patient with Dr. Eugene E. Record and Dr. Barr. The question was whether the operation that had already been carried out had cured her, because it would be a source of profound distress to find no tumor whatsoever in a hindquarter resection. Dr. Barr, however, had undertaken a careful reconstruction after resection of an area of bone disease on the assumption or in the hope that the lesion was benign. The attempt to deal in that way with tumors has been supported by Phemister* and others in an attempt to remove areas only rather than to sacrifice an entire limb; the experience has been deplorable in that the malignant tumors almost invariably recur. Technically, the hindquarter amputation was a simple procedure in this young person. She withstood it very well and was able to go home shortly after operation.

The interesting thing is that of all the hindquarter amputations that we have done here more

*Phemister, D. B. Personal communication.

than half have been done for chondrosarcoma of the head and neck of the femur and the adjacent innominate bone, which are the favorite sites for this tumor; they are the ones that have the most favorable prognosis for radical surgery because they tend to metastasize late and leisurely, and there is a reasonable hope that the tumor tissue will be removed before metastases have occurred. All the previous chondrosarcomas in this area that we have had have been very bulky tumors with extensive, discernible soft-tissue masses that caused marked distortion. Dr. Aufranc and I together saw one that was nearly as large as a football and completely filled the pelvis, pushing the organs out of the pelvis and crushing them against the opposite side. The present tumor is the only one that I have seen that had not bulged out with an enormous soft-tissue mass. I think it is not surprising that the diagnosis was not even seriously contemplated until the time of operation.

DR. AUFRANC: Was the acetabulum fractured, or was there any involvement beyond the bone?

DR. KAHN: No, it was a central chondrosarcoma limited to the body and neck of the femur. We did not find a fracture in the acetabulum.

DR. AUFRANC: The x-ray films led me to believe that it extended beyond the acetabular margin.

DR. TAYLOR: You also pointed out changes in the innominate bone rather remote from the acetabulum. I suppose you have to say that they were due to disuse atrophy.

DR. AUFRANC: I did not mean to imply that those changes were lesions, but I did believe that the tumor had gone across the joint.

DR. KAHN: It was limited to the femur. The bones of the pelvis showed only some osteoporosis.

DR. LEIDHOLT: Is there any way to account for the marked synovial reaction other than to say that metastases had occurred into the synovial tissue? Certainly, none were seen macroscopically. Did any additional sections show tumor?

DR. KAHN: We made multiple sections of the synovia, none of which showed tumor. I think the synovial reaction was secondary to the change in the mechanics of the hip joint.

DR. LEIDHOLT: The change was due to the fracture and the distortion of the femoral head.

CASE 39202

PRESENTATION OF CASE

A forty-seven-year-old woman was admitted to the Emergency Ward because of hemoptysis.

The patient was too weak to give a complete history, so the husband furnished the details. Three years before admission insidious weight gain, exertional dyspnea and fatigue had developed. A basal metabolic rate was reported as normal. Since that time she had had severe exertional dyspnea accompanying even slight activity, orthopnea requiring four pillows for relief, paroxysmal nocturnal dyspnea and palpitation. Seven months before admission she had heart failure with ankle swelling and was treated at another hospital with mercurial diuretics and digitalis. No mercurial diuretics had been taken for the last four months, and ankle swelling had not recurred. She had continued to take digitalis daily. Sixteen months, seven months and five months before admission she had bouts of "pneumonia," treated at another hospital. She had night sweats but no chills. Four months before admission she coughed up small amounts of blood-streaked sputum for three days and was again admitted to the other hospital, where tuberculosis was supposedly ruled out. Three months later "grippe" had developed, and she had been in bed since. She had lost 38 pounds in the last two weeks. On the evening of admission sharp, right-sided, parasternal chest pain, penetrating to the back and aggravated by breathing and coughing, developed and was accompanied by the coughing up of large amounts of clotted blood and bright-red, frothy sputum.

She denied ever having had scarlet fever, rheumatic fever, chorea or arthritis. However, since early adulthood she had had "heart disease," said not to be rheumatic, but "just a large heart." Five years before admission she had had pneumonia. Two and a half years before admission she had been in an automobile accident in which she was struck in the chest by her husband's elbow but no definite injury had occurred.

Physical examination revealed a thin, pale woman. There was cyanosis of the lips and fingers. She was not co-operative and tolerated examination poorly. The neck veins were distended and pulsating at 45°. The apex of the heart was within the left midclavicular line. The rhythm was regular, the sounds were of good quality, and there were no murmurs. The aortic second sound was greater than the pulmonic. In the right side of the chest below the scapula there were flatness, absent breath sounds and absent tactile fremitus anteriorly and posteriorly. There were coarse moist rales over the right axilla and right anterior apex. The liver edge was palpable one fingerbreadth below the lower costal border and was somewhat tender. There was no peripheral edema.

The temperature was 104°F., the pulse 120, and the respirations 28. The blood pressure was 120 systolic and 80 diastolic.

A specimen of the urine was not obtained. Examination of the blood showed a hemoglobin of 9.5 gm. per 100 cc. and a white-cell count of 30,200, with 78 per cent neutrophils, 14 per cent lymphocytes and 8 per cent monocytes.

A roentgenogram of the chest demonstrated an upper dorsal scoliosis convex to the left. There was crowding of the ribs on the right side. The

trachea appeared to be displaced to the right, partly because of the thoracic deformity. The lower half of the right-lung field was opaque, suggesting atelectasis of the middle and lower lobes. Within the opaque area there was a crescentic translucency consistent with the appearance of air in a cavity (Fig. 1). The right intermediate bronchus appeared to be displaced upward. On the left side the left pulmonary artery and its main branches

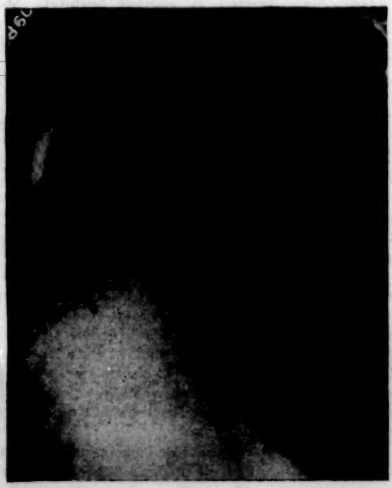

FIGURE 1. *Roentgenogram of the Chest.*
The arrow points to the crescentic air shadow. Note also the displacement of the heart and mediastinal structures.

appeared considerably enlarged. The distal vessels were relatively small.

Morphine, Mercuhydrin, Digalen and crystacillin were given, and she was placed in an oxygen tent. The severe paroxysms of coughing slowed and she became more comfortable. Watery mucus was aspirated from the throat by nasal catheter. However, breathing slowed and the patient died quietly twelve hours after admission.

DIFFERENTIAL DIAGNOSIS

DR. ARTHUR S. PIER, JR.*: I wonder if I could see the x-ray films.

DR. JOHN F. GIBBONS: We have just one set of chest roentgenograms, which were taken on admission. There is a large upper dorsal scoliosis, convex to the left, slight displacement of the trachea to the right side, perhaps aggravated by the scoliosis, displacement of the heart and mediastinal structures to the right side and obliteration of the lower right half of the wrist; within this obliteration there is a crescentic shadow of air density, which suggests that this may be air within a cavity (Fig. 1). The shift of the mediastinum and the apparent elevation of the diaphragm on that side suggest collapse of the lower lobe and perhaps of the middle lobe. The intermediate bronchus, which should come off the right main bronchus, may be uplifted; it seems to be pursuing a horizontal course although that is not a definite finding. On the left side the main pulmonary artery and its branches are dilated, and although the summary states that the peripheral vessels are small I think that they are of good size too, as can be seen in the lateral view. The left lung is otherwise clear.

DR. PIER: Can you tell us anything about the left heart border?

DR. GIBBONS: The left border appears to be normal.

DR. PIER: Do you think the aorta is somewhat small?

DR. GIBBONS: I can only say that the aortic arch is not very prominent; therefore, the aorta presumably is not dilated to any significant degree.

DR. PIER: It seems to me that there are three questions about this patient that we must try to answer. What caused her death? What was the process seen in the right lung in the x-ray film? And what was the nature of the heart disease, if any?

For the terminal episode, during which she had severe parasternal pain aggravated by breathing and coughing and raised large amounts of blood, it seems to me the most likely explanation is a pulmonary embolism and infarction. There are, I suppose, other possibilities one would casually consider. I thought vaguely of a dissecting aneurysm, but there was no history of hypertension, the coughing up of such large amounts of blood would be unusual, and the picture of the left pulmonary artery is to me much more suggestive of pulmonary embolism than dissecting aneurysm. When a sudden sharp pain develops near the sternum, one wonders about mediastinal emphysema and rupture of the lung, but the air seen in the roentgenogram was not close to the mediastinum and there was no systolic crunch on physical examination. Furthermore, I do not think mediastinal emphysema would account for the large amount of blood raised. I shall call the terminal episode a large pulmonary embolus associated with or accentuating right-sided heart failure. Certainly, when the patient came into the hospital she had pulsating neck veins and a slightly low liver edge and probably did have right-sided heart failure.

Passing on to the question of the process in the right lung, I thought at first that I might explain the whole thing on the basis of changes that followed the pulmonary embolus. An embolus,

*Assistant in medicine, Massachusetts General Hospital.

though, would not produce these definite x-ray changes within a few hours; therefore, some process was probably present for quite a long time. The patient had bouts of pneumonia and had raised small amounts of bloody sputum in the past; she had had the so-called "grippe" for at least a month. Moreover, on admission she had a temperature of 104°F. and a white-cell count of 30,000, both of which, although perfectly compatible with a pulmonary embolus, occurred too soon after the terminal episode to be caused entirely by that embolus. The air in the lung field is certainly suggestive of a cavity so that she may have had a lung abscess. I believe that she had a lung abscess with pneumonitis around it and a good deal of pulmonary collapse. Why should she have had this pulmonary difficulty? It is possible that she had had numerous episodes of pulmonary infarction — that these episodes of so-called pneumonia were really pulmonary infarcts. Perhaps the period of "grippe" was really a series of small infarcts. Although it is possible that she had had an old pulmonary infarct in the right lung that became infected, there may well have been in the beginning a pneumonia that failed to resolve and led to an abscess. It looks as though there was some element of bronchial obstruction to cause the apparent collapse and the persistent infection in that lung; that again raises the question, What was the cause of the bronchial obstruction in this patient? I do not think the history is particularly suggestive of a bronchogenic tumor. I suppose she could have had an adenoma, but that means dragging in another diagnosis and I should prefer to leave it out.

I shall go on to the nature of the heart disease. The patient was said to have had a large heart since early adult life, but was not supposed to have had rheumatic fever. There was no history of hypertension. Could it have been a congenital heart disease? I think the most likely congenital lesion would be a large defect of the atrial septum. If such a defect produces a large pulmonary artery, it is often associated with a small aorta and not infrequently ends in heart failure or pulmonary emboli; it is usually accompanied by a harsh pulmonic systolic murmur. No murmur was mentioned here, but if she had an embolus in the pulmonary artery it may have altered the murmur or obliterated it, I suppose. I think defect of the atrial septum is a far-fetched possibility. Since there was a three-year period of heart failure with exertional dyspnea, palpitations, orthopnea and blood spitting, the most likely diagnosis is rheumatic heart disease. I have always believed, rightly or wrongly, that on rare occasions a tight mitral stenosis may produce no murmur; certainly the murmur of mitral stenosis is variable and frequently drops out entirely. This patient was undoubtedly orthopneic, and in the sitting position a murmur could easily have been missed. The fact that the heart sounds were of good quality led me to think that she might well have had mitral stenosis because with mitral stenosis the first sound at the apex is abrupt and loud and gives the impression of a heart with good, vigorous action. The pulmonic second sound was less loud than the aortic, which would be unusual in mitral stenosis, but perhaps the pulmonary embolus diminished — the right side had perhaps failed so that the sound had become less loud than it would ordinarily have been. I should think that a tight mitral stenosis leading to a large right atrium could cause enough bronchial obstruction — certainly some distortion and upward displacement of the bronchi — to allow pulmonary infection to take place.

I shall say for my diagnosis that the patient died of a large pulmonary embolus, that she had an abscess in the right lung, with pneumonitis and collapse, and that basically she had rheumatic heart disease with a tight mitral stenosis.

DR. BENJAMIN CASTLEMAN: Do you think the left atrium is enlarged?

DR. GIBBONS: I cannot say with any certainty that it is.

DR. ALLEN G. BRAILEY: How are the shadows of the left arteries? I cannot see them from here.

DR. GIBBONS: They are extremely dilated pulmonary vessels.

DR. HUGH R. DUDLEY, JR.: I followed this patient; it was only after she died that we received the x-ray report describing the crescentic translucency. We had previously been given an x-ray description of thrombosis of the left pulmonary artery so that we were also impressed with the clearness of the left-lung field and the prominence of the left pulmonary artery. We thought that she had some pneumonia because of the fever and the elevated white-cell count.

DR. EDWARD B. BENEDICT: I do not quite see how this would explain the whole picture. There is so much evidence of pulmonary destruction that I do not think we can explain it without a bronchial adenoma or a slowly growing carcinoma.

DR. PIER: I think that the patient may well have had obstruction; I think a large heart might do this.

DR. HELEN S. PITTMAN: I do not see how a large heart would do this. It seems to me that one must postulate collapse on the right side to explain this complete radiodensity, the physical finding of absent breath sounds and the displacement of the heart to the right. There must have been a collapsed lung in that area, with a closed bronchus. I also should think that the patient had thrombosis of the left pulmonary artery and repeated episodes of infarction, but I wonder if the so-called pneumonitis was not merely occlusion of the bronchus, with absorption of the air trapped behind it.

DR. J. T. HEYL: Do you ever see pulmonary thrombosis progressing retrogradely?

Dr. Castleman: Yes; it may begin in the lung and extend toward the hilus, because of superimposed thrombosis.

Clinical Diagnosis

Pulmonary embolism.
Cor pulmonale.
?Pneumonitis.

Dr. Arthur S. Pier, Jr.'s Diagnoses

Abscess in right lung, with pneumonitis and collapse.
Pulmonary embolism.
Rheumatic heart disease, with mitral stenosis.

Anatomical Diagnoses

Chronic organizing pneumonitis, right lower lobe, with abscess formation.
Bronchopneumonia.
Cor pulmonale (heart weight, 425 gm.), with focal endocardial fibrosis, right ventricle.
Pulmonary-artery dilatation and arteriosclerosis.

Pathological Discussion

Dr. Castleman: Autopsy showed that the right lower lobe and most of the right middle lobe were almost entirely solid. In the upper portion of the right lower lobe was an abscess cavity, 4 to 5 cm. in diameter and filled with necrotic material and blood, that communicated with a large bronchus. The abscess was surrounded by solid lung, which proved to be an organized pneumonia. I believe that this patient at one time had a pneumonia of the right lower lobe — she was supposed to have had pneumonia five years before admission — and that later she had three or four attacks of pneumonia that never resolved, and that in the area of organization an abscess cavity developed. There was a great deal of fibrosis; the alveoli, atria and respiratory bronchioles were filled with organized exudate. The infection had produced many enlarged regional lymph nodes, which accounted for the elevation of the bronchus seen on the roentgenogram. There were several large nodes in the carinal angle and all along the bronchi. We toyed with the idea that the abscess represented a sequestration in the right lower lobe but were unable to confirm it. There was a bronchial communication, and there was no aberrant artery coming from the aorta. An extensive terminal bronchopneumonia was noted in the rest of the lung.

The heart was enlarged, weighing 425 gm., but most of the enlargement was dilatation and hypertrophy of the right ventricle, which measured twice the normal thickness and was probably twice the size of the left ventricle. We were unable to find any congenital abnormality. The endocardium of the right ventricle was whitish and slightly thickened such as we often see with regurgitation, but there was no evidence that there had been any regurgitation. To go along with the hypertrophy of the right ventricle was a marked dilatation of both pulmonary arteries with severe atherosclerosis, indicating that there had been hypertension in the pulmonary circuit for a long time. This dilatation extended far out to the periphery, and the pulmonary arteries measuring as little as 3 mm. in diameter showed large atheromatous plaques. We were hoping to find in some of the smaller vessels evidence of emboli such as we have seen on occasion that might answer the whole problem but were unable to find them. There was certainly hypertension in this circuit, but we have no explanation for it. To be sure, the patient had a scoliosis and a moderate amount of emphysema, but usually when that produces right-sided hypertrophy, there is not marked dilatation of the pulmonary vessels and not as much evidence of hypertension in the pulmonary circuit. I think we have to leave it as one of the cases with pulmonary hypertension, marked dilatation of the pulmonary artery and atherosclerosis without thrombosis, cause unknown.

Dr. Pittman: Was the organized pneumonia accompanied by enough vascular fibrosis to obliterate pulmonary vessels and to give rise to the pulmonary hypertension?

Dr. Castleman: I do not think so.

Dr. Gordon S. Myers: I should like to add that it is possible to remove one lung by pneumonectomy so that only half the pulmonary vascular bed remains without creating pulmonary hypertension. It has been pointed out that inflammatory lesions of the lung may give rise to striking collateral circulation from the bronchial artery and that in turn could have some effect on raising the pressure in the pulmonary system, but that would not explain the picture here. It seems that this patient must have had severe pulmonary hypertension for many years, and it seems equally unlikely that the process in the lung was present for that long a period.

Dr. Pier: What was the cause of the pain?

Dr. Castleman: There was an acute fibrinous pleuritis.

Dr. Pier: The dyspnea over the period of a year is interesting.

Dr. Castleman: The patient had right-sided heart failure in the past, and she had evidence of that at autopsy in the form of a marked central necrosis of the liver.

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"EACH NEW TEMPLE"

THE Boston Medical Library, now inaugurating its long-considered and carefully planned campaign for funds with which to carry on its vital functions in the community, may rightly be classed among the country's great repositories of learning. Its antecedents go back even farther than its seventy-eight years of uninterrupted existence, for what might be called a trial run of twenty-one years was started in 1805 when the first organization of this name came into being. When this early institution was liquidated in 1826 its literary assets went to the Boston Athenaeum, a process that was reversed years later on the turning over of the medical contents of the Boston Public Library to the present Medical Library.

This library, founded in 1875 under the presidency of Oliver Wendell Holmes, had as its first home two rented rooms in Hamilton Place. In three years it had outgrown these quarters and purchased the house of Dr. Samuel Gridley Howe in Boylston Place. In 1900, as a result of the generosity of Mrs. Warren B. Potter and others, the present building rose at 8 The Fenway; the space provided served the institution's purposes until 1930, when it became necessary to add the still unfinished "new" wing, one floor of which is occupied by the *Journal* and which contains in addition the austere, still unplastered periodical room, two floors of modern steel stacks and above them a vast undeveloped space with wooden book boxes clinging to its walls like swallows' nests. It is this space that now calls urgently for completion in order that it may house in orderly fashion the piles of books and pamphlets that are heaped throughout the rest of the building.

During these years of quiet growth the Library has maintained its place as third largest in the country, exceeded only by the Armed Forces Medical Library — formerly the Library of the Surgeon General of the Army — and that of the New York Academy of Medicine. Its position is assured by the possession of 218,634 volumes and 155,642 pamphlets, all catalogued, in addition to thousands of still unprocessed items, of which 71,149 were put into circulation in 1952.

Probably few users of the smaller peripheral libraries of New England, both medical and otherwise, realize that in their excursions into medical literature they are regularly tapping the great central reservoir on the edge of Boston's Fens. By the interlibrary loan system public libraries and the working libraries of hospitals and medical schools are constantly drawing on these almost inexhaustible resources. A Veterans Administration hospital truck, for example, averages two calls a day to the Library, and other hospital and medical-school conveyances make several calls each week.

When, some five years ago, the Massachusetts Medical Society accepted a large share of the year-by-year support of the Library on a membership basis it committed itself to a mature and far-seeing policy in the patronage of medical culture. In

this respect it must stand nearly alone among state societies, for having accepted responsibility for a library whose usefulness extends far beyond the Society's membership; for the promotion of a post-graduate teaching institute that places no geographical limitations on those whom it will serve, and in the publication of a far-traveling journal.

Now that the Library must expand in order to continue its indispensable functions, it is a foregone conclusion that a medical profession wise in the knowledge of its own functions will give its eager support. It is not too much to expect that many thoughtful lay persons, encouraged by the medical counsel that they hold valuable, will see, in this need of the Library for a quarter of a million dollars to expand its facilities and increase its service, a basic need of the community that defies appraisal.

One can do no better than to recall certain lines written by the Library's first president:

> — Leave thy low-vaulted past!
> Let each new temple, nobler than the last,
> Shut thee from heaven with a dome more vast. . . .

COMMISSIONER OF PUBLIC HEALTH

THE appointment of Dr. Samuel B. Kirkwood as commissioner of public health for Massachusetts represents the recognition of an able and versatile physician whose background of medical service, teaching and research is a testimonial to competency and professional standing.

Dr. Kirkwood evidenced an early interest in public health when, after graduation cum laude from Harvard Medical School in 1931 and internships in pathology and surgery, he became epidemiologist with the Division of Tuberculosis of the Massachusetts Department of Health for the year 1934-35 under Dr. Alton S. Pope. His next five years were spent in internships, residencies and research (pathology) at Boston hospitals in the field of obstetrics and gynecology, an experience that became the basis of his faculty connection in 1939 with the Harvard Medical School and later in the Department of Maternal and Child Health of Harvard School of Public Health. The social responsibilities of his clinical and public-health interests were strengthened by his appointment in 1941 as chief of staff of the Florence Crittenton Maternity Home.

The war extended Dr. Kirkwood's steadily enlarging experience, and in 1942 he was commissioned in the Army Medical Corps, serving for two years in Army hospitals before specializing in military preventive medicine. In preparation for this work he attended the School of Military Government at the University of Virginia late in 1944 and the Civil Affairs Training School at Yale University from November, 1944, to May, 1945. After duty with the Civil Affairs Staging Area, Presidio of Monterey, California, he was named public-health officer, Headquarters Staff, of the Army's Fifth Amphibious Corps in Japan in September, 1945. There he took charge of the Military-Civilian Health Control Program on the Island of Kyushu. In January, 1946, he was assigned as public-health officer to I Corps Headquarters in Japan. In this position Dr. Kirkwood's primary job was to bring under control typhus and smallpox epidemics then plaguing Japanese civilians in the Corps area, and to cope with the threat occasioned by the return of soldiers harboring malaria, dysentery and other diseases affecting public health.

After returning to the United States, Dr. Kirkwood was commissioned a colonel in the United States Army Reserve Medical Corps and has shown himself one of the most active and interested of officers in his appreciation of the need for continued military preparedness during the present critical period of world unrest. Since returning to this country, he has resumed his duties as assistant professor of maternal health at Harvard School of Public Health and has maintained clinical interests that should make him, as Massachusetts Commissioner of Health, unusually sympathetic to the problems of the practitioners and their patients as well as to health organizations and the rest of the community that is aiming to avoid disease.

The Governor is to be congratulated on his appointment of a physician who is a man of mature stature and demonstrated professional attainment — one who knows both sides of the controversies that can arise between those whose main business it is to cure people and those whose efforts are primarily devoted to controlling the very hazards that keep

their colleagues busiest. A forward outlook, a balanced temperament and a broad experience are great assets to a health commissioner, and Massachusetts is fortunate in having had a long line of such physicians. The present incumbent gives promise of upholding this tradition.

ETIOLOGY AND EPIDEMIC CHARACTER OF ACUTE GLOMERULONEPHRITIS

THE relation of acute glomerulonephritis to streptococcal infections has long been recognized, but the reasons for its irregular occurrence in patients with such infections have not been understood. At the 1952 meeting of the Association of American Physicians, Rammelkamp, Weaver and Dingle[1] called attention to an important difference between rheumatic fever and acute glomerulonephritis in this respect. They pointed out that the incidence of rheumatic fever bears a relatively constant relation to that of streptococcal infections both at different times in any given area and in widely separated localities. By contrast, whatever information is available points to an irregular attack rate for acute nephritis.

In attempting to track down possible reasons for the difference, these workers studied the streptococci associated with initial attacks of acute glomerulonephritis. Among 12 strains from as many sporadic cases obtained in 5 widely scattered areas, 10 proved to belong to Type 12 and 2 to Type 4; in addition 4 siblings of 3 of the patients who yielded the Type 12 strains had evidence of acute nephritis, but cultures from these patients were not available. This finding was not a coincidence related to the unusual prevalence of Type 12 streptococci. Only one of the Type 12 strains came from the only recognized case of acute nephritis observed at the Air Force base in Cheyenne where studies of streptococcal infections have been conducted for the past several years; during this time there were only 42 untreated patients with Type 12 streptococcal infections, as compared with 1164 with infections due to streptococci of other types. A number of additional, small circumscribed epidemics of acute nephritis were noted as having been studied recently and found to be associated with Type 12 streptococci.

Further pursuing their findings Rammelkamp and Weaver[2] were able to uncover a total of 31 cases of acute nephritis in four years in various cities in this country; among them Type 12 streptococci were associated with 26 attacks, Type 4 streptococci with 4 others, and Type 25 with 1. Type 18 streptococci were isolated from 2 cases in an outbreak of nephritis in Hawaii. These findings were interpreted as demonstrating that infection with Type 12 streptococci and perhaps with Type 4 strains are particularly apt to be followed by an attack of acute nephritis. Considerable evidence was presented from various sources that strains of Type 12 streptococci may vary in nephritogenic capacity.

Corroborative evidence of the relation of Type 12 streptococci to acute nephritis was presented in a retrospective study by Wertheim and his co-workers.[3] They had isolated Group A hemolytic streptococci from 39 patients with acute nephritis during the seven-year period, 1936-42; 20 of these were identified as Type 12, and the remainder as a variety of other types. Since these strains were obtained at various intervals after the onset of the nephritis, some of them may have represented new types introduced after the appearance of the initial infection. These and other technical problems of isolating and identifying streptococci make the finding of Type 12 strains in such a large proportion of cases more impressive, and the results tend to support the observations of Rammelkamp and his co-workers.

These findings may be of more than casual interest. In the first place, they suggest that identification of streptococci may help to uncover subclinical cases of acute nephritis, particularly among close contacts of known cases. This has already been accomplished by clinicians and epidemiologists who provided Rammelkamp with some of his material. Also, since there is evidence that immunity in hemolytic streptococcal infections may be type-specific, infection with Type 12 streptococci may offer protection against subsequent recurrences of acute nephritis. Moreover, there already is evidence strongly suggesting that the administration of penicillin to patients with Type 12 streptococcal infections may prevent the subsequent development of acute nephritis, but gamma globulin fails

to do so.[4] These observations offer the prospect of preventing the spread of nephritis within family groups or other population units by identification of cases and of carriers of specific nephritogenic types and application of the best available methods for eradicating the streptococci from such persons.

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WHO . . . ME?

Under the above title and with appropriate humorous if somewhat grimly suggestive illustrations, the Public Information Department of The Travelers Insurance Companies of Hartford introduces the nineteenth edition of its series of highway safety booklets. First published in 1931 these brief but pointed pamphlets have presented almost annually their statistical data and pithy comments on the year's traffic toll and some of the reasons for it.

"Who . . . . Me?" obviously represents the surprised reaction of the average careless, stupid or deliberately dangerous driver to the implication that he might be a logical candidate for participation in a highway disaster.

The statistics for 1952 are not encouraging in this respect. Traffic deaths in the United States reached a total of 37,600, — an increase of 500 over the previous year's record, — and injuries on the highway reached the appalling number of 2,090,000, more than 127,000 over the 1951 mark. Each of these figures for 1952 is the highest in the history of the world since it came to be inhabited by the motor vehicle.

An analysis of the data is interesting and perhaps in some ways contrary to expectation. Of the fatalities, 14,970 resulted from collision between automobiles, 8,650 from collision with pedestrians, 3,570 from collision with fixed objects and 1,580 from violent encounters with railroad trains. Other types of accident accounted for 8,120 deaths. The stage was set with clear weather when 26,280 of the fatal accidents occurred, and the roads were dry when 24,490 of them took place. Moreover, 30,720 of the deaths occurred on *straight roads*, bearing out the observation that excessive speed is the commonest cause of motor-vehicle accidents. In this respect the modern superhighway has proved to be the Jehu's undoing. Built for speed, it encourages speed, and speed is intrinsically dangerous. Though highways are equipped with every safety device to reduce this intrinsic hazard, it is impossible to eliminate the fact that each highly mobile engine of destruction is in the hands of a single vulnerable human being.

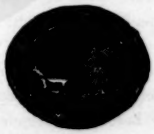
Over 17 per cent of the accidents occurred in the dismal hours before cockcrow — between 1 a.m. and 6 a.m.; nearly 25 per cent of them involved drivers under twenty-five years of age, although this age group constitutes only about 15 per cent of all drivers. Youth must have its fling and does not count the penalty. Over 98 per cent of the drivers involved had had a year or more experience in driving. Familiarity, apparently, still breeds contempt.

And so the question "Who . . . Me?" is finally answered: "Yes, YOU!"

A Boston lady has at this time a somewhat novel disease — a continual motion of the tongue, which no device, effort of the will, or medication, controls.

Boston M. & S. J., *May 18, 1853*

MASSACHUSETTS MEDICAL SOCIETY



DEATH

Harvey — Clifford D. Harvey, M.D., of Brookline, died on April 23. He was in his seventy-fourth year.

Dr. Harvey received his degree from Hahnemann Medical College and Hospital of Philadelphia in 1910. He was formerly associate professor of surgery at Boston University School of Medicine and was a member of the staffs of Massachusetts Memorial Hospitals, Leonard Morse Hospital, Natick, Morrison Hospital, Whitefield, New Hampshire, and Huggins Hospital, Wolfeboro, New Hampshire. He was a fellow of the American College of Surgeons and American Medical Association.

He is survived by his widow, two sons and a daughter.

CIRCULAR OF ADVANCE INFORMATION

It has been urged by members of the Council of the Massachusetts Medical Society and the Executive Committee of the Council that the *Circular of Advance Information for Councilors*, which contains reports from the committees of the Society, be made available to all fellows, in order that they may consider them and keep their district councilors informed concerning their views.

To supply every fellow of the Society with this circular would cost a considerable sum. Consequently, it will not be sent out until it is known how widespread the demand may be.

Copies will be sent to all fellows who ask for them as long as the present supply lasts. Future requests for copies should be made before August 1, December 1 or March 15 for the Council meetings of October, May and February respectively. If a sufficient number of requests are received, copies will be made available for all fellows.

ROBERT W. BUCK, M.D.
Secretary pro tempore

CORRESPONDENCE

CURE OF PLANTAR WARTS BY INJECTION

To the Editor: The impression is seemingly conveyed by the article on the local injection treatment of plantar warts by Branson and Rhea and by the editorial regarding it, both of which appeared in the *Journal* of April 9, that this form of therapy is new and original. As a matter of fact, this particular mode of treatment for plantar warts has been known and practiced for at least twenty years.

In 1932 I began the local injection of plantar warts with novocain and soluble bismuth solutions. The novocain was used for its anesthetic effect since it was realized with the very first case that the patient would withdraw his foot and thus prevent the introduction of the bismuth into the portion of the wart intended — namely, its base. About 1.5 cc. of novocain solution was injected subcutaneously into the tissues adjacent to and below the wart. After local anesthesia had been attained, an additional 0.5 cc. was introduced hypodermically into the base of the wart itself, the correct location being ascertained by the fact that pressure had to be exerted in the introduction of the solution. Five to 8 minims of a soluble bismuth preparation, 1.5 per cent aqueous solution of bismuth sodium tartrate was then injected obliquely into the base of the wart after the needle (26 gauge) enters the skin just lateral to the visible hyperkeratotic portion of the wart. The use of bismuth was based on the fact that cases had previously been reported in which its parenteral (intramuscular) administration had effected the disappearance of verrucae in locations distant from the area of injection. It was believed, moreover, that the locally injected bismuth might have a sclerosing effect on the many small blood vessels as a rule present in a wart.

Up to 1934 I treated 5 cases in this manner. The plantar wart or warts disappeared after one treatment in 4 cases and after two treatments in the fifth. In the last the wart had recurred two months after its disappearance. All these cases had resisted previous excision or x-ray therapy. Mention or report of these results was precluded, however, by the report of Shallow on the "Treatment of Verrucae by Local Injections of Bismuth," which appeared that year (*Illinois Medical Journal* 66:332-336, 1934). Treating 67 cases of painful palmar and plantar warts he attained cure in 64, improvement in 2 and failure in 1 with one to three local injections of a 1.5 per cent aqueous solution of bismuth sodium tartrate. No mention was made of the employment of novocain in his report, although it is extremely difficult to realize how it is possible to introduce the desired medicament (bismuth) into the base of a plantar wart without prior local anesthesia. On the basis of my previous experience and on that of Shallow, I have continued the local injection of plantar warts with novocain and soluble bismuth solution until recently, with excellent clinical results. Numerically, the cases are essentially similar to his and far superior to those obtained by Branson and Rhea, indicating the important place of bismuth in this special therapy.

It must be emphasized that it is a wart that is to be injected and not a callus or a corn, in which the treatment is absolutely valueless.

J. L. GRUND, M.D.

Brookline, Massachusetts

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Lehrbuch der Geburtshilfe. By Prof. Dr. Heinrich Martius, director, Universitäts-Frauenklinik, Göttingen. With the assistance of Prof. Dr. Werner Bickenbach, director, Universitäts-Frauenklinik, Tübingen. Illustrated by Käthe Droysen. Second revised edition. 4°, cloth, 770 pp., with 786 illustrations. Stuttgart: Georg Thieme, 1952. Ganzl. DM 58.80. (Distributed by Grune and Stratton, New York.)

This new edition of a book first published in 1948 has been completely revised and brought up to date. The text is divided into chapters on pregnancy, birth, multiple births, the puerperium, lactation, the newborn and hygiene of the mother. The publishing, and especially the illustrative work, is excellent. The book should be in obstetric collections in large medical libraries, and should prove of value to German-reading physicians.

Physics and Medicine of the Upper Atmosphere: A study of the aeropause. The Proceedings of a Symposium on the Physics and Medicine of the Upper Atmosphere held at San Antonio, Texas, November 6, 7, 8, 9, 1951. Sponsored by The Air University School of Aviation Medicine, Randolph Field, Texas. Edited by Clayton S. White, M.D., director of research, Lovelace Foundation for Medical Education and Research, and head, Section of Aviation Medicine, Lovelace Clinic; and Otis O. Benson, Jr., brigadier general, USAF (MC), and commandant, USAF School of Aviation Medicine. 4°, cloth, 611 pp., with 149 illustrations and 46 plates. Albuquerque: University of New Mexico Press, 1952. $10.00. (Arranged by The Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico.)

Thirty-eight physicians and scientists participated in this symposium on the upper atmosphere. All phases of the subject were discussed, including travel by man in the upper regions of the air and the aeropause. Lists of references are appended to the various chapters, and there is a good index. An atlas of forty-six plates follows the text. The publishing is excellent and the book is recommended for all collections on aviation medicine.

Methods of Medical Research. Vol. 5. A. C. Corcoran, editor-in-chief. 8°, cloth, 394 pp., with illustrations. Chicago: Year Book Publishers, Incorporated, 1952. $7.50.

This new volume in a valuable reference series is the joint work of 56 specialists in particular fields. The subjects covered include methods for separation of complex mixtures and substances of higher molecular weight, technics of renal study and immunochemical procedures for determining homogeneity of proteins and polysaccharides. Lists of references are appended to the chapters, and there are indexes of authors and subjects. The set of volumes is recommended for all medical libraries.

Living with Cancer. By Edna Kaehele. 12°, cloth, 160 pp. Garden City, New York: Doubleday and Company, Incorporated, 1952. $2.00.

This is the true story of a woman who learned to live and survive with a malignant cancer.

Rypins' Medical Licensure Examinations: Topical summaries and questions. By Walter L. Bierring, M.D., secretary, Federation of State Medical Boards of the United States, chairman, American Board of Preventive Medicine and Public Health, and Iowa State Commissioner of Health. With the collaboration of a review panel. Seventh edition. 8°, cloth, 856 pp. Philadelphia: J. B. Lippincott Company, 1952. $8.00.

This invaluable reference work has been revised and brought up to date by the editor, assisted by a panel of 10 outstanding teachers and clinicians. There is a comprehensive index, and the publishing is excellent in every way. The book is recommended for all medical libraries.

Angst und Schrek in klinisch-psychologischer und sozialmedizinischer Sicht: Dargestellt an Hand von Erlebnisberichten aus dem Luftkrieg. By Prof. Dr. Friedrich Panse, chief physician, Hirnverletzten-Instituts des Landes Nordrhein-Westfalen, Bonn. 8°, paper, 189 pp., with 4 illustrations. Stuttgart: Georg Thieme, 1952. DM 12.60. (*Arbeit und Gesundheit*, New Series, No. 47.)

Dr. Panse discusses anxiety and terror in their clinical, psychologic and medicosociologic aspects. The coverage is comprehensive in its scope, with an evaluation of the important literature. There is a bibliography of four pages.

Laboratory Technic in Biology and Medicine. By E. V. Cowdry, research professor of anatomy and director, Wernse Cancer Research Laboratory, Washington University, St. Louis. Third edition. 8°, cloth, 382 pp. Baltimore: Williams and Wilkins, 1952. $4.00.

This new edition of a standard work has been thoroughly revised and brought up to date. It is actually the joint effort of 70 specialists, who describe their special technics. The material is arranged alphabetically. The book should be in all reference collections in medical libraries and should prove valuable to laboratory technicians.

Sammlung versorgungs-und gerichtsärztlicher Gutachten aus dem Gebiete der inneren Medizin. By Dr. Med. Heinz Meyeringh, division physician for internal diseases and chief physician, Observation Division, Städt. Krankenhauses Priwall, Lübeck-Travemünde, and legal physician, chief Insurance Office, Schleswig. 8°, paper, 188 pp. Stuttgart: Georg Thieme, 1952. DM 14.70. (Distributed by Grune and Stratton, New York — *Arbeit und Gesundheit*, New Series, No. 47.)

The author discusses the medicolegal opinions concerning various disorders of the organs and systems of the human body as exemplified by cases from official records.

Physicians' Handbook. By Marcus A. Krupp, M.D., assistant clinical professor of medicine, Stanford University School of Medicine, and director, Palo Alto Medical Research Foundation; Norman J. Sweet, M.D., assistant professor of medicine, University of California School of Medicine; Ernest Jawetz, Ph.D., M.D., associate professor of bacteriology and lecturer in medicine and pediatrics, University of California School of Medicine; and Charles D. Armstrong, M.D., clinical instructor in medicine, Stanford University School of Medicine. Seventh edition. 16°, cloth, 380 pp., with illustrations. Los Altos, California: Lange Medical Publications, 1952. $2.50.

This new edition of a popular handbook, first published in 1941, has been thoroughly revised, and some of the sections have been expanded.

Research in Endocrinology. By August A. Werner, M.D., and associates. Edited by Al R. Schmidt, city editor, *Belleville Daily Advocate*, Belleville, Illinois. 8°, cloth, 285 pp., with illustrations and tables.

The first part of this small book has to do with researches of Dr. Werner and his associates over a period of twenty-six years of teaching at St. Louis University School of Medicine. It contains lists of his writings, and lectures and exhibits, and a short biography. The larger part of the book comprises a collection of sixteen important reprints collected from various sources and brought together for easy reference purposes.

Essentials of Dermatology. By Norman Tobias, M.D., associate clinical professor of dermatology, St. Louis University School of Medicine, assistant dermatologist, Firmin Desloge and St. Mary's hospitals, visiting dermatologist, St. Louis State Hospital, and visiting physician in the department of dermatology, St. Louis Hospital. Fourth edition. 12°, cloth, 596 pp., with 186 illustrations and 3 colored plates. Philadelphia: J. B. Lippincott Company, 1952. $6.00. Lippincott Essentials Series.

This new edition of a manual first published in 1941 has been thoroughly revised in the light of advances in therapy, pathology and etiology. The material on the virus diseases, the erythrodermas and the allergic and collagen disorders has been expanded.

Diseases of the Ear, Nose and Throat: Principles and practice of otorhinolaryngology: clinical technics and procedures. By Francis L. Lederer, M.D., professor and head of the department of laryngology, rhinology and otology, University of Illinois College of Medicine, chief, Otolaryngological Service, Research and Educational Hospital, captain, medical corps, United States Naval Reserve, and director of otolaryngology and chief, Ear, Nose and Throat Service, Illinois Eye and Ear Infirmary. Sixth edition. 4°, cloth, 1430 pp., with 979 illustrations. Philadelphia: F. A. Davis Company, 1952. $20.00.

Dr. Lederer has extensively revised this new edition of his book, first published in 1938. There are new chapters on allergy, general pathology, ophthalmology, psychiatry, roentgenology, and speech and voice. The book has been greatly expanded, with the main objective of providing an encyclopedia on otolaryngology. The format is two column, and there is a comprehensive index. The volume should be in all collections on the subject.

Medicine and Pathology. Edited by V. Zachary Cope, M.D., M.S. (Lond.), F.R.C.S. (Eng.). With a general preface to the series by the editor-in-chief, Sir Arthur S. MacNalty, K.C.B., M.D., F.R.C.P., F.R.C.S. 8°, cloth, 565 pp. London: Her Majesty's Stationery Office, 1952. 50s. net. (*Medical History of the Second World War, United Kingdom Medical Series.*)

This is the first volume of the British official medical history of World War II. It is proposed to complete the series in nine sections. The present volume is the first of two on clinical medicine and surgery. It comprises thirty-two chapters, written by different persons, on general and special medicine. Lists of references are appended to the chapters, and there is a good index. A valuable list of previous official medical histories of war precedes the text. The publishing is excellent. The set should be in all collections on military medicine and surgery.

Rorschach's Test: III. Advances in Interpretation. By Samuel J. Beck, Ph.D., Institute for Psychosomatic and Psychiatric Research, Michael Reese Hospital, Chicago, and Department of Psychology, University of Chicago and Northwestern University. 8°, cloth, 301 pp., with 9 tables. New York: Grune and Stratton, 1952. $5.50.

The second volume of this series was published in 1945. This new volume is based on researches carried on by the author during the past five years. Four cases are given in detail. A list of references and indexes of authors and subjects are appended to the text.

The Pathogenesis and Treatment of Thrombosis: With a clinical and laboratory guide to anticoagulant therapy. By Irving S. Wright, M.D., professor of clinical medicine, Cornell University Medical College. 8°, cloth, 78 pp., with 27 illustrations and 1 table. New York: Grune and Stratton, 1952. $3.00. (*Modern Medical Monographs No. 1.*)

Dr. Wright, in this small monograph, discusses the modern concepts of thrombosis. Lists of references are appended to the chapters, and there is a good index.

Veterans Administration Technical Bulletins: Series 10, Volume V, 1951. 4°, cloth, 165 pp., with 14 illustrations. Washington, D. C.: United States Government Printing Office, 1952.

This volume contains eleven papers on clinical subjects, including one on sarcoidosis by Dr. Warfield T. Longcope.

Electrotherapy and Actinotherapy: A textbook for student physiotherapists. By E. B. Clayton, M.B., B.Ch. (Cantab.), consulting physician, Physical Treatment Department, King's College Hospital, London. Second edition. 8°, cloth, 452 pp., with 127 illustrations. London: Baillière, Tindall and Cox, 1952. $4.00.

The first edition of this special book was published in 1949. This new edition has been thoroughly revised. Material has been added on valve-generated muscle-stimulating currents. There is a good index, and the publishing is excellent.

Gynäkologische Strahlentherapie: Indikationsstellung, Methodik und ärztliche Betreuung. By Dr. Med. Richard K. Kepp, professor of obstetrics and gynecology, University of Göttingen. With an introduction by Prof. Dr. Heinrich Martius, director, Universitäts-Frauenklinik, Göttingen. 8°, cloth, 232 pp., with 64 illustrations. Stuttgart: Georg Thieme, 1952. DM 26. — (Distributed by Grune and Stratton, New York.)

This monograph on radiotherapy of the diseases of women is divided into two main sections: nonmalignant and malignant disorders. The last section, which discusses the malignant tumors of the female organs, including the breast, comprises the greater portion of the text, 152 pages. There is a bibliography of 22 pages and a good index. The publishing is excellent, as it is with all Thieme books. The monograph should be in collections on radiotherapy and gynecology.

Die Frühdiagnose des Uteruscarcinoms: Histologie, Kolposkopie, Cytologie, biochemische Methoden. By Prof. Dr. Med. Hans Limburg, chief physician, Universitäts-Frauenklinik, Hamburg. Second revised edition. 8°, cloth, 208 pp., with 83 illustrations. Stuttgart: Georg Thieme, 1952. DM 19.50. (Distributed by Grune and Stratton, New York.)

This second edition of an important monograph has been revised and brought up to date. It is illustrated with a large number of histologic slides, and there is a bibliography of 10 pages. The monograph should be in collections on cancer and gynecology.

Der Tuberkuloseablauf im Körper: Grundlagen der phthisiogenetischen Analyse. By Reiner W. Müller, Köln. 8°, cloth, 170 pp., with 55 illustrations. Stuttgart: Georg Thieme, 1952. DM 24. — (Distributed by Grune & Stratton, New York.)

Dr. Müller has written a valuable monograph on the pathogenesis of tuberculosis, surveying the essential literature on the subject. A bibliography of 6 pages concludes the text, and there is a good index. The publishing is excellent. The book should be in all collections on tuberculosis.

Estudos Cirúrgicos. 6. Série. Eurico B. Ribeio, director, Sanatorio São Lucas. 8°, cloth, 226 pp., with illustrations. São Paulo: Editôra S. A., 1952.

This sixth volume of a series of surgical case reports covers the whole field of surgery. There is also an index for the complete series of six volumes.

Rheumatische Erkrankungen: Entstehung und Behandlung. Edited by Prof. Dr. Max Hochrein, physician-in-chief, Medizinischen Klinik, Ludwigshafen am Rhein. Second revised edition. 8°, cloth, 416 pp., with 121 illustrations. Stuttgart: Georg Thieme, 1952. DM 36. — (Distributed by Grune and Stratton, New York.)

This second edition of a collective work written by a number of specialists has been thoroughly revised, bringing the knowledge on the rheumatic diseases up to date. The coverage is comprehensive, all aspects of the disease and its relation to the various organs and systems of the body being included. There are chapters on the disease as a social problem, its occurrence in children and women and its relation to the atmosphere and as an occupational hazard. Eighty-five pages are devoted to the recognition and treatment of infectious rheumatism. There is a bibliography of 23 pages and a good index. The book is recommended for all collections on the subject.

Anatomie und Physiologie: Lehrbuch für ärztliches Hilfspersonal. By Professor Dr. Med. Joseph Bücker, Hamburg. Seventh revised edition. 8°, cloth, 152 pp., with 90 illustrations. Stuttgart: Georg Thieme, 1952. DM 15.60. (Distributed by Grune and Stratton, New York.)

This manual for technicians has enjoyed an unusual popularity. First published in 1939, it has gone through seven editions. This new edition has been revised, and material added on vitamins, hormones and metabolism. The text, which is well organized, is divided into two main sections, general and special. The former is devoted to tissues and the structures of passive movement. The latter discusses in order the locomotive system and muscles, the blood and circulatory system, the respiratory, digestive, genitourinary and nervous systems, the organs of sense and the skin. There is a good index, and the publishing is excellent in every way, especially the illustrative work. The book should prove valuable to German-reading personnel.

Le indicazioni ed i metodi della resezione epatica. By Corrado Cavina, instructor in surgical anatomy and operative surgery, Università di Perugia, associate surgeon, Ospedal Maggiore di Bologna, and primary titular surgeon, Ospedale Civile di Fabriano. 8°, paper, 110 pp., with 32 illustrations. Bologna: Licioni Cappelli-editore, 1952. L. 1000.

Dr. Cavina discusses the indications and methods of resection of the liver. The preliminary chapters, which are devoted to the history of the subject, are followed by those on preoperative exploration, indications and contraindications, and operative methods. There is a bibliography of 6 pages.

Lavori dell'Istituto di Anatomia e Istologia Patologica della Università degli Studi di Perugia. Vol. 12, Part 1, 1952. 4°, paper, 144 pp., with illustrations. Perugia: Tip. "Grafica" di Salvi & Co., 1952.

This number of a periodical from an important Italian institute contains articles on mast cells in carcinoma of the stomach and hypophysis, streptomycin in tuberculosis, neoplasms of the breast, cyst of the parotid gland and statistics of biopsies.

Biologie d'anopheles Gambiae: recherches en Afrique-Occidentale Française. By M. H. Holstein, Dr. ès. Sc., in charge of research, Office de la Recherche scientifique Outre-Mer, Paris. 8°, paper, 176 pp., with 22 illustrations and 31 tables. Genève: Organization Mondiale de la Santé, 1952. (*Série de Monographies*, No. 9.) $2.00.

This monograph discusses the morphology, the biology of the first stage and of the adult, the races and varieties of *Anopheles gambiae* and its role in the transmission of malaria.

Physiologie normale et pathologique du métabolisme de l'eau: déshydratations, oedèmes déséquilibres hydriques. By Jean Hamburger, assistant professor, Faculté Médecin des Hôpitaux de Paris; and Georges Mathé, Médaille d'Or intern, Hôpitaux de Paris. 8°, cloth, 502 pp., with 53 illustrations and 25 tables, and 10 plates. Paris: Éditions Médicales Flammarion, 1952. 2850 francs.

In this monograph the authors present a comprehensive survey of the existing knowledge of the normal physiology and pathology of the metabolism of water in the human body. The material is divided into three large sections on physiology, methods of measurement and pathology. There is a bibliography of 2536 titles on which the analysis of the authors is based. There is a good index, and the publishing is excellent. The price in francs seems appalling but reduced to dollars amounts to about $8.50. The book is a valuable reference tool and should be in all collections on metabolism.

Das Problem der Schizophrenie: Analytische Psychotherapie und Psychose. By Dr. Med. Harald Schultz-Hencke. 8°, cloth, 308 pp. Stuttgart: Georg Thieme, 1952. DM 27. (Distributed by Grune and Stratton, New York.)

This monograph on schizophrenia briefly discusses the subject in general. There is an extensive analysis of a single case. A large part of the text, 72 pages, is devoted to history of the various theories of the disease from Griesinger (1845) to Schilder (1925). The monograph should be in all collections on mental disorders.

Cardiac Therapy. By Harold J. Stewart, M.D., associate professor of medicine, Cornell University Medical College, attending physician, New York Hospital, and head, Division of Cardiology, Department of Medicine, New York Hospital–Cornell University Medical Center. 4°, cloth, 622 pp., with 68 illustrations and 11 tables. New York, New York: Paul B. Hoeber, Incorporated, 1952. $10.00.

This book, as its title indicates, is devoted to the treatment of diseases of the heart and cardiac complications in association with other diseases. There are chapters on heart disease in old age and in pregnancy, cardiac management of surgical patients, surgery in cardiac conditions and diet and prevention. The coverage is extensive, and there is a comprehensive index. The volume is too heavy for its size, owing to the paper used in printing. A lighter paper could be used to advantage if a second edition is issued at a later date. The publishing is otherwise excellent.

Gynecologic and Obstetric Pathology with Clinical and Endocrine Relations. By Emil Novak, M.D., D.Sc. (Hon. Trinity College, Dublin). Third edition. 8°, cloth, 595 pp., with 630 illustrations. Philadelphia: W. B. Saunders Company, 1952. $10.00.

Dr. Novak has thoroughly revised this new edition of his well known textbook and has added considerable material, including a chapter on diseases of the breast. Nearly a hundred new illustrations have been incorporated in the text, and the illustrative work is excellent. The references appended to the chapters have been brought up to date; there is a good index. The book should be in all gynecologic and obstetric collections.

Pharmaceutical Calculations. By Willis T. Bradley, A.M., Carroll B. Gustafson, Ph.C., S.B., A.M., and Mitchell J. Stoklosa, Ph.C., S.B., A.M., of the faculty of the Massachusetts College of Pharmacy, Boston. Second edition. 12°, cloth, 290 pp. Philadelphia: Lea and Febiger, 1952. $3.75.

The authors of this new edition of a special manual have added much material throughout the text. Sample practice problems have been appended to the various chapters, with the answers concluding the text. It is poor practice to print tables on the inside of the covers of any book. Otherwise, the publishing is good. The small book should prove valuable to medical librarians for reference purposes.

Rheumatic Fever: A symposium held at the University of Minnesota on November 29, 30, and December 1, 1951, under the sponsorship of the Minnesota Heart Association. Edited by Lewis Thomas, M.D., American Legion Memorial Heart Research Professor, departments of Pediatrics and Medicine, University of Minnesota Medical School. 8°, cloth, 349 pp., with 105 illustrations. Minneapolis: University of Minnesota Press, 1952. $10.00.

Twenty-four papers, covering all the aspects of the subject, were read at the three-day symposium. The references for all the papers are combined in a 21-page bibliography, which concludes the text. The lack of an index is noted. The book should be in all collections on the subject.

Viral and Rickettsial Infections of Man. By Thomas M. Rivers, M.D., director of the Hospital, Rockefeller Institute for Medical Research. Second edition. 4°, cloth, 719 pp., with 90 illustrations, including 7 plates in color. Philadelphia: J. B. Lippincott Company, 1952. $7.50.

This new edition of an authoritative reference book has been thoroughly revised and brought up to date. The book is the joint work of 30 specialists in their particular fields. There are new chapters on the human reactions produced by the Coxsackie virus, hemagglutination by viruses and interference between viruses. Material on epidemic viral gastroenteritis, cat-scratch disease and generalized salivary-gland virus infections has been added. Lists of references are appended to the chapters. There are indexes of authors and subjects, and the publishing is excellent. The price is low for the type of book. The volume should be in all medical libraries.

NONCLINICAL NOTES

LEONARD WOOD MEDAL

Dr. David D. Scannell was last month the fifth recipient of the Leonard Wood medal, marking a career of distinguished service to the Boston City Hospital. He had served the institution from 1903, the year he joined the Massachusetts Medical Society, to 1936, when he retired after five years as surgeon-in-chief.

NOTICES

MOUNT AUBURN HOSPITAL LECTURES

The last in the series of public medical lectures sponsored by the Mount Auburn Hospital will be presented in Margaret Jewett Hall, 330 Mount Auburn Street, Cambridge, on Wednesday, May 20, at 8:15 p.m. Dr. R. Cannon Eley will speak on the subject "Preventable Diseases in Infancy and Childhood."

Admission is free.

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

The New England Society of Physical Medicine will meet at the luncheon of the Section on Physical Medicine of the Massachusetts Medical Society at the Hotel Statler, Boston, on Tuesday, May 19, at 12 noon. Howard A. Rusk, M.D., professor and chairman, Department of Physical Medicine and Rehabilitation, New York University College of Medicine, will speak on the subject "Rehabilitation."

Luncheon tickets must be purchased in advance.

ASSOCIATED DIPLOMATES OF THE NATIONAL BOARD OF MEDICAL EXAMINERS

The annual meeting of the Associated Diplomates of the National Board of Medical Examiners will be held at the Belmont-Plaza Hotel, Forty-Ninth Street and Lexington Avenue, New York City, on Wednesday, June 3, at 3:30 p.m.

KAPPA DELTA AWARD FOR RESEARCH IN ORTHOPEDIC SURGERY

A prize of $1000, donated by the Kappa Delta Sorority, may be awarded annually by the American Academy of Orthopaedic Surgeons for the best research in orthopedic surgery performed by an American citizen in the United States. The selection will be made from work published since January 1, 1951, or research presented to the Committee on Scientific Investigation of the American Academy of Orthopaedic Surgeons before November 1, 1953.

Further information may be obtained from Dr. John J. Fahey, 1791 West Howard Street, Chicago 26, Illinois, chairman of the Committee on Scientific Investigation of the American Academy of Orthopaedic Surgeons.

SOCIETY MEETINGS AND CONFERENCES

MAY 18–22. National Tuberculosis Association. Page 664, issue of April 9.

MAY 19. New England Society of Physical Medicine. Notice above.

MAY 19–21. Massachusetts Medical Society. Hotel Statler, Boston.

MAY 20. Massach..setts Medico-Legal Society. Hotel Statler, Boston. 2:30 p. m.

MAY 20. Mount Auburn Hospital Lecture. Notice above.

MAY 20. Massachusetts School Physicians' Association. Page 663, issue of April 9.

MAY 25–31. First World Congress on Fertility and Sterility. Page 1001, issue of December 18.

MAY 28–31. American Therapeutic Society. Page 746, issue of April 23.

MAY 30 AND 31. Society for Investigative Dermatology, Inc. Page 570, issue of March 26.

JUNE 3. Associated Diplomates of the National Board of Medical Examiners. Notice above.

(Notices concluded on page xix)